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# DISEASES

OF THE

# Stomach, Intestines,

AND

# Pancreas

BY

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WITH 388 ILLUSTRATIONS, SOME IN COLORS

*SECOND EDITION, REVISED AND ENLARGED*

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TO

**William H. Thomson, M. D., LL.D.,**

CONSULTING PHYSICIAN TO THE ROOSEVELT AND RED
CROSS HOSPITALS AND LATE PRESIDENT
OF THE NEW YORK ACADEMY
OF MEDICINE,
THIS VOLUME IS INSCRIBED
AS A TRIBUTE TO HIS HIGH PROFESSIONAL ATTAINMENTS AND IN REMEMBRANCE OF HIS
MANY ACTS OF KINDNESS,
BY THE AUTHOR

# PREFACE TO THE SECOND EDITION

In placing the second edition of his work on Diseases of the Stomach and Intestines before the medical profession the author has endeavored, as far as possible, to avail himself of the criticisms of his former book and to correct those errors which were called to his attention. There are several new subjects which have been introduced, notably chapters on Colon Bacillus Infection and on Diseases of the Pancreas. The section on Duodenal Ulcer has been rewritten. It has been the writer's endeavor to furnish the general practitioner with a working knoweldge of the subject, and to select for him from the great accumulation of material the most practical methods.

Many physicians have neither the time nor the opportunity to devote to a clinical course on these subjects, and next in value to this for the purpose of instruction is the employment of photography to demonstrate the methods of diagnosis and of treatment. Of this I have endeavored to take advantage. Many of the illustrations are from photographs of patients at the Manhattan State Hospital, taken by Mr. Hill, the official photographer, for whose services I am indebted to the courtesy of Dr. Wm. Mabon, the medical superintendent. Some of the illustrations have been made by my artist, Mr. Thomas Nast, Jr., from models.

As visceral displacements have assumed an important position, their symptoms, diagnosis, and treatment, notably by mechanical methods, are specially described. It has been my endeavor to clearly set forth the indications for surgical procedure, and to demonstrate particularly the futility of medical treatment in surgical conditions, such as in benign stenosis of the pylorus, and the necessity of early exploration for the purpose of diagnosis in suspected carcinoma of the stomach. These merely serve as illustrations.

The author frankly advocates Russell Chittenden's views as to the danger of excessive protein diet, and from personal clinical experience is the more convinced of the soundness of Chittenden's theory.

Typhoid fever is again included in this volume on account of its intestinal complications and for the purpose of differential diagnosis. In the chapter on this subject the writer particularly discusses the question of diet and, in his belief, the excessive calorie values which have been advocated. There is a chapter devoted to Diverticulitis.

Pure gastric neuroses the author believes to be rather rare, and particular attention is directed to reflex gastro-intestinal disturbances emanating from disease of the gall-bladder, appendix, and other organs.

ROBERT COLEMAN KEMP.

NEW YORK CITY, *August*, 1912.

# PREFACE

In view of the excellent works on diseases of the stomach and intestines that have been placed before the medical profession, the publication of a new book on these subjects might almost seem to be superfluous. From a great accumulation of material, it is often difficult for the general practitioner to select simple and practical methods, and it is the endeavor that this volume should render service in this special direction.

Many physicians have neither time nor opportunity to devote to a practical clinical course, and next in value to this for the purpose of instruction is the employment of photography to demonstrate the methods of diagnosis and treatment. Of this I have endeavored to take advantage. Many of the illustrations are from photographs of patients at the Manhattan State Hospital, taken by Mr. Hill, the official photographer, for whose services I am indebted to the courtesy of Dr. Wm. Mabon, the Medical Superintendent. Some of the illustrations have been made by my artist from models.

As visceral displacements have recently assumed an important position, their symptoms, diagnosis, and treatment, notably by mechanical methods, are specially described. Typhoid fever is included in this volume on account of its intestinal complications and for the purpose of differential diagnosis.

A chapter is devoted to *Diverticulitis*, which has become an important subject.

The endeavor has been made to indicate as clearly as possible the conditions which call for surgical procedure.

ROBERT COLEMAN KEMP.

NEW YORK CITY.

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# DISEASES

OF THE

# STOMACH, INTESTINES, AND PANCREAS

## PART I

## CHAPTER I

## ANATOMY OF THE STOMACH AND INTESTINES

### ANATOMY OF THE STOMACH

A BRIEF description will be given of the anatomy of the stomach, but for a complete exposition of the subject the reader is referred to any standard anatomy.

The stomach lies in the epigastric and left hypochondriac regions, about five-sixths of it to the left of the median line. The larger end, the fundus, fits into the concave left vault of the diaphragm. Modern investigation by means of the *x*-rays has demonstrated that the stomach does not lie transversely across the abdomen, as was formerly described. The fundus is nearly vertical, and the pyloric portion nearly transverse—this is, of course, with the patient in the standing posture. The position may be described as ⅃ shaped, that is, a reversed L (Fig. 1). It may be well compared to a gourd.

The cardiac orifice (*C*), the junction of the esophagus and stomach (the esophageal orifice), is fixed and lies behind or a little to the left of the sternal junction of the left seventh cartilage (seventh rib), or about 1¼ inches from the edge of the sternum, in the left parasternal line, on a level with the spinous process of the ninth dorsal vertebra. The cardia is situated 4½ inches from the anterior surface of the abdomen. The point of communication with the small intestine is called the pylorus (*P*), and shows a furrow on the outer surface and within a protruding fold (the valve of the pylorus).

The pylorus (*P*) lies between the right sternal and parasternal lines, slightly below the tip of the ensiform process, and corresponds to the spinous process of the twelfth dorsal vertebra. It descends slightly when the stomach is distended and moves somewhat to the right. A line (+), drawn in the axis of the esophagus through the stomach to its lower border, cuts off about one-fourth of the organ to the left. This portion is called the greater cul-de-sac or fundus (*F*).

The fundus (*F*) rises as high as the lower border of the left fifth rib in the mammillary line, slightly above and behind the apex of the heart, and from 1 to 2 inches higher than the cardia. It is in contact with the diaphragm above, and to the left with the spleen and left kidney.

The lesser curvature (*L*) lies to the left of the vertebral column, passes downward and parallel with it, and then crosses it to the upper border of the pylorus.

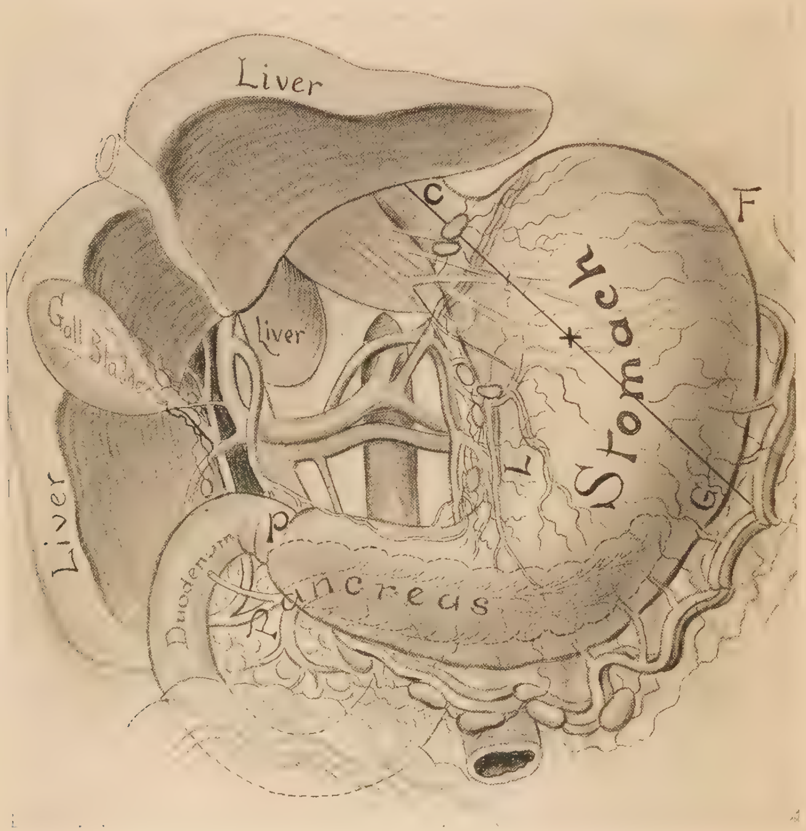

Fig. 1.—The stomach: *C*, Cardia; *F*, fundus; *P*, pylorus; *L*, lesser curvature; *G*, greater curvature (modified from W. J. Mayo).

The greater curvature (*G*) forms the fundus and lower border of the stomach and extends to the lower border of the pylorus. The lower border, when the organ is distended, lies about two to three fingers' breadth ($1\frac{1}{2}$ to $2\frac{1}{4}$ inches) above the umbilicus.

The volume of the stomach varies according to its contents. Dehio has shown that the healthy stomach when empty is contracted and hidden away in the left cavity of the diaphragm, and it is the colon that we then demonstrate by percussion.

The pancreas and splenic vessels lie behind the stomach. The anterior surface is overlapped above by the liver, the left lung, and the seventh, eighth, and ninth ribs. Below it is in relation with the abdominal wall.

The pyloric end, the lesser curvature, and the cardia lie behind and beneath the quadrate and left lobes of the liver.

Traube's space is the area in which the stomach lies in direct contact with the ribs, and is bounded above by the liver and left lung, externally by the spleen, and the inner border is formed by the free costal margin. Both here and in the epigastric region pure gastric tympany can be elicited.

When the stomach is distended, the lesser curvature is directed obliquely backward toward the spine, the posterior wall looking somewhat downward and the anterior wall slightly upward. The transverse colon, if distended, may overlap the greater curvature, and the latter tends to fall away from the abdominal wall when the patient is in the dorsal position. The transverse colon lies ordinarily below the greater curvature. With moderate distention, the average length from fundus to pylorus is 10 to 12 inches; from the lesser to greater curvature, 4 to 5 inches; from the anterior to the posterior wall, about 3 to 3½ inches.

The average capacity is variable. It may contain even as much as 2 quarts. A plane drawn transversely through the base of the lesser curvature will lie parallel with the plane of the diaphragm.

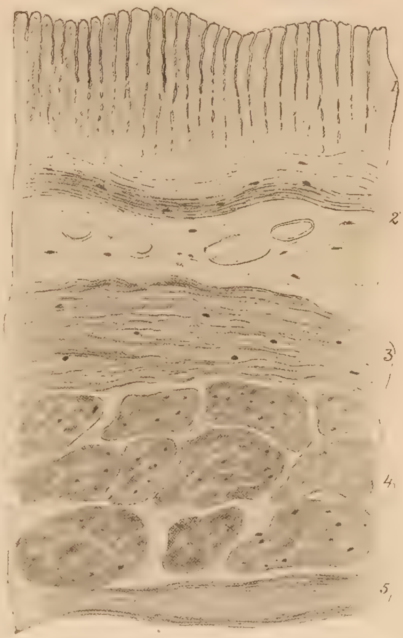

Fig. 2.—Vertical section of the stomach: 1, Mucosa; 2, submucosa; 3, 4, muscularis; 5, serosa.

The lesser omentum extends from the lesser curvature to the liver above, and the great omentum is suspended from the greater curvature, protecting the viscera.

The blood-vessels enter the upper and lower borders, and thus divide the surface into two equal parts. They mark the greater and lesser curvatures.

**Structure of the Stomach.**—The stomach consists of four coats: serous or peritoneal, muscular, submucous or areolar, and mucous. The peritoneal coat forms a thin, transparent, elastic membrane, and closely covers the organ, except along the curvatures, where it is more loosely attached for the passage of the blood-vessels.

The muscular coat consists of three sets of fibers (Fig. 2), disposed in layers—the outer or longitudinal, middle or circular, and inner or oblique. The last is a continuation of the circular fibers of the esophagus, and the fibers descend obliquely from the cardia upon the anterior and posterior surface, and, spreading out like a fan, terminate at the greater curvature.

The submucous coat consists of areolar tissue, connecting the mucous and muscular coats. The blood-vessels subdivide therein.

The mucous membrane is soft, smooth, somewhat pulpy, and of pink color, thickest in the pyloric region and thinnest at the fundus. It constitutes the glandular layer of the organ. It is covered by columnar epithelial cells, which extend for a variable distance into the mouths of the glands. There are about five million glands in the stomach, tubular in form, and perpendicular to the surface. They are surrounded by fibrous tissue and lymphoid cells and by a thin muscle layer (muscularis mucosæ).

The glands are composed of a mouth, neck, body, and base, and several tubules, from two to even four or five, may end in one mouth. On microscopic examination, the dots appearing on the surface of the mucosa are the openings of the glands. There are three varieties of glands in the stomach:

Cardiac or fundus glands; pyloric glands; mucous glands.

*Cardiac* or *fundus glands* are the most numerous. They fill the greater part of the stomach, and are characterized by the shortness of their mouths and the length of the glands. They contain two varieties of cells. Cells bordering on the lumen of the tube, which are small, granular, and polyhedral or columnar, the chief or principal cells, and which stain only to a slight extent with anilin dyes. The other cells, parietal or oxyntic, lie between the principal cells and the membrana propria (Fig. 3). They are most numerous in the necks of the glands, larger than the chief cells, are oval or angular, and finely granular in structure. They have strong affinity for anilin dyes.

*Pyloric Glands.*—These are characterized by the greater length of their mouths, which are lined by cylindric epithelium. They are found only in the region of the pylorus. The body or secretory portion of the gland is represented by a single layer of short and finely granular columnar cells, resembling the chief cells of the fundus glands. There are also a few isolated cells (Nussbaum) which resemble, in structure and in their behavior to anilin dyes, the parietal cells of the fundus glands (Fig. 4).

Besides these specific glands, a number of *mucous glands* are found near the pylorus.

The hydrochloric acid is secreted by the parietal cells: pepsin and the milk-curdling ferment by the principal cells of the fundus and pyloric glands. Some consider the mucus to be also a product of the cylindric goblet-cells lining the stomach and the wider portions of the glandular ducts. The ferments do not exist as such in the cells, but as zymogens, which are transformed into ferments through the activity of the free hydrochloric acid.

*Blood-vessels, Lymphatics, and Nerves of the Stomach.*—The *arteries* of the stomach are derived from branches of the celiac axis, the gastric and pyloric branch of the hepatic artery supplying the upper curvature, and forming the superior ventricular arch, and the right gastro-epiploic from the hepatic, and the left gastro-epiploic and vasa brevia from the splenic, forming the inferior ventricular arch.

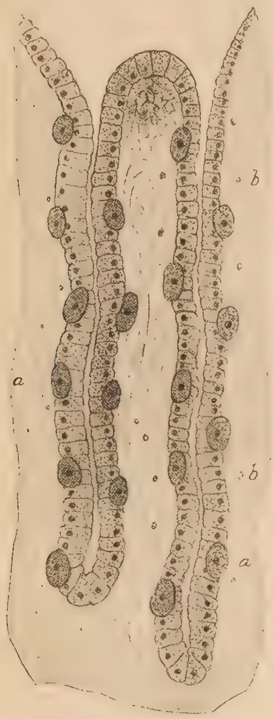

Fig. 3.—Cardiac gland: *a*, Parietal cells; *b*, principal cells.

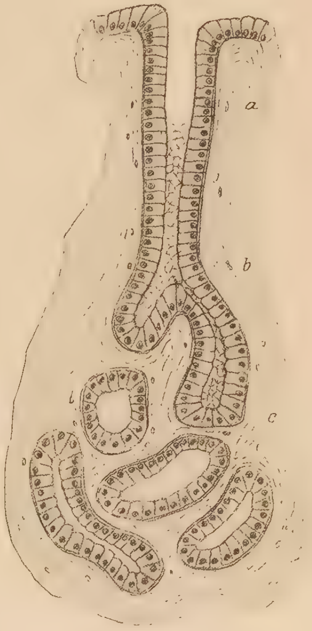

Fig. 4.—Pyloric gland: *a*, Mouth; *b*, neck; *c*, fundus.

They reach the stomach between the folds of peritoneum and ramify between the muscular coats, giving off a number of capillaries and dividing into small vessels in the submucosa, and finally enter the mucous membrane and pass between the tubuli, where they form a plexus of fine capillaries both on the walls of the tubules and around the mouths of the glands.

The *veins* arise from this capillary network and pass nearly straight through the mucous membrane between the glands. They pierce the muscularis mucosæ and form a plexus in the submucosa, and finally form the coronary and pyloric veins emptying into the portal vein, the right gastro-epiploic vein emptying into the superior mesenteric vein, and the left gastro-epiploic vein emptying into the splenic vein.

The *lymphatics* extend directly to the surface of the mucosa. They form a dense network of lacunar spaces between and among the gland tubuli, which they inclose, as well as the blood-vessels, with sinus-like dilatations. The lymph is collected near the surface of the mucous membrane into vessels which form loops and possess dilated extremities. They are less superficial than the capillaries, though the lacunar spaces extend as far as the basement membrane of the surface. The lymphatic glands extend *along the lesser curvature of the cardia*, while they are present on the greater curvature from the *pylorus to only about one-fourth to one-third of the distance.* This fact is of important consideration in gastrectomy for carcinoma, as the lesser curvature must, therefore, be entirely removed.

*Nerves.*—The left vagus supplies the anterior surface of the stomach. The right vagus supplies the posterior surface with only one-third of its fibers, the remainder passing to the other viscera.

Branches of the sympathetic nerves pass from the celiac plexus and anastomose with the vagi. These nerves, with numerous ganglia, form a network in the submucosa.

## ANATOMY OF THE INTESTINES

The intestinal canal is divided into two parts: the small intestine and the large intestine; the former about 7.6 meters (25 feet) long, and the latter 1.5 to 1.8 meters (5 to 6 feet) long.

The small intestine is subdivided into three portions,—the duodenum, jejunum, and ileum,—and lies, excepting the duodenum, to the inner side of the large intestine, and is connected to the posterior abdominal wall by the mesentery, which last incloses the jejunum and ileum throughout.

**The Duodenum.**—The duodenum, which is about 10 to 12 inches (25.5–30.5 cm.) long, is not suspended by the mesentery, and is the most fixed and widest part of the small intestine, having a diameter of 1½ to 2 inches (3.81–5.08 cm.). It is curved like a horseshoe, surrounds the pancreas, and is divided into four parts (Fig. 5).

The superior horizontal part of the duodenum is about 2 inches (5.08 cm.) long, begins at the pylorus at the level of the first lumbar vertebra, and passes slightly upward and to the right of the gall-bladder. It is the most movable portion, is surrounded by the peritoneum, and suspended chiefly by ligaments from the hilus of the liver and neck of the gall-bladder. The quadrate lobe and neck of the gall-bladder lie above it; below it is the pancreas, and behind it the common bile-duct and hepatic vessels.

The descending portion of the duodenum, about 3 inches (7.5 cm.) long, commences at the neck of the gall-bladder and runs vertically to the third or fourth lumbar vertebra on the right side, and touches the right kidney. The transverse colon passes in front of it; on the left side is the pancreas, and the common bile-duct lies a little posterior. At its inner and back part, about 4 inches from the pylorus, the common bile-duct and pancreatic duct enter it and form the diverticulum or ampulla of Vater, and the duct of Santorini enters it a short distance above.

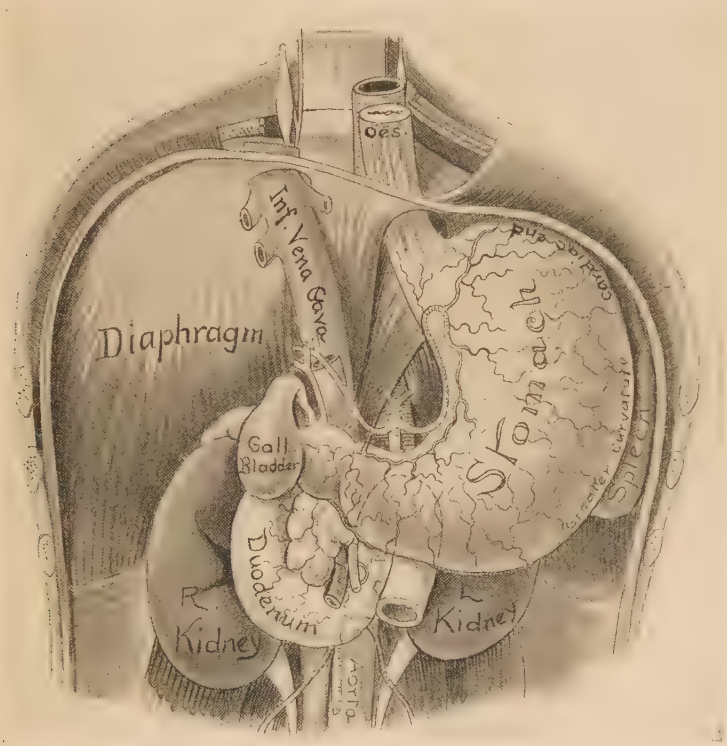

Fig. 5.—Stomach and duodenum, liver and intestines removed, and showing anatomic relations of the duodenum (after Testut).

The third or transverse portion of the duodenum, about 5 inches (12.5 cm.) long, extends from the right side of the body of the third or fourth lumbar vertebra across the spine, and slightly ascends to the left side of the spine. The superior mesenteric vessels cross it, as does the mesentery. The lower layer of transverse mesocolon lies in front. The pancreas and superior mesenteric artery lie above, and the aorta, vena cava, and crura of the diaphragm behind it. It is the most fixed portion of the duodenum.

The fourth or ascending portion of the duodenum, about 1 to 2 inches (2.54–5.08 cm.) long, ascends vertically along the left side of the spine, from the third or fourth lumbar vertebra to the side of the second or first lumbar vertebra. It is firmly fixed by the suspensory muscle of the duodenum (muscle of Treitz), which descends from the left crus of the diaphragm. Anteriorly are the transverse colon and transverse mesocolon (lower layer). It terminates in the jejunum (usually opposite the second lumbar vertebra) and forms the duodenojejunal flexure.

**Jejunum and Ileum.**—They form the continuation of the duodenum. It is hard to determine where the one ends and the other begins. The jejunum occupies the upper two-fifths of the remaining small intestines, or about 9 feet 7 inches (2.9 meters); the ileum, the lower three-fifths, or about 14 feet 5 inches (4.3 meters), and ends at the ileocecal junction. Their position is not fixed, but the jejunum is more apt to occupy the left side of the abdominal cavity, with the loops more transverse; while the ileum is usually found on the right side and in the pelvis, with the loops more vertical.

The coils of the jejunum and ileum are very movable and are completely invested by peritoneum. They are supported and attached to the posterior parietes by the mesentery, which is attached above to the left of the vertebræ on a level with the lower border of the pancreas. The mesentery runs downward and to the right and presents the form of a fan, the intestines hanging on it in the form of coils.

The diameter of the jejunum is about 1½ inches (3.81 cm.), and this gradually diminishes in size to the ileum; and in this, in turn, the diameter decreases until its entrance into the large intestine. The ileum passes nearly perpendicularly into the ascending colon, its mucosa forming a double valve (valvula Bauhini).

The jejunum and ileum are the most movable parts of the intestinal tract. They are often met with in hernias, and if pregnancy, a tumor, or ascites are present, the intestines move up and escape compression.

Occasionally Meckel's diverticulum (the remains of the vitelline duct), a process 2 or 3 inches long, is given off from the ileum, on an average of 1 to 2 feet above the ileocecal junction. It is of importance in reference to intestinal obstruction.

**Arterial Supply of Small Intestine.**—The duodenum is supplied by the pyloric branch of the hepatic, by the superior pancreaticoduodenal branch of the gastroduodenal branch of the hepatic, and by the inferior pancreaticoduodenal branch of the superior mesenteric, and the jejunum and ileum by the superior mesenteric artery. They branch into small vessels which run through the intestinal wall, ramify in the submucosa, and form the capillary system of the villi and glands.

**Veins.**—The venous blood flows partly into the superior gastric vein and partly into the superior mesenteric vein, and empties into the vena porta.

**Lymphatics.**—The lymphatics are divided into those of the mucous membrane and muscular coat, and form plexuses. They run between the folds of the mesentery and end in the mesenteric lacteals, and so on into the intestinal lymphatic trunk and thoracic duct. They are provided with valves to prevent a backward flow.

**Nerves.**—The duodenum is supplied by the hepatic plexus, a branch of the celiac plexus, with branches of the right vagus.

The superior mesenteric plexus, formed by nerves from the celiac plexus, the semilunar ganglia, and right vagus, supply the jejunum and ileum.

The nerves enter the intestinal wall with the blood-vessels and form a subserous net. They then penetrate the longitudinal muscular fibers and form between these and the circular muscular fibers ramifications consisting of numerous groups of multipolar cells (Auerbach's plexus), from which fine branches supply the muscular tissue. Other branches penetrate the circular muscular layer to the submucosa, where they form the submucous nerve plexus (Meissner's plexus), and branches supply the muscularis mucosæ, the muscles of the villi, and end in the mucosa.

**Structure of the Small Intestine.**—The small intestine is composed of four coats: serous or peritoneal, muscular, submucous, and mucous (Fig. 6).

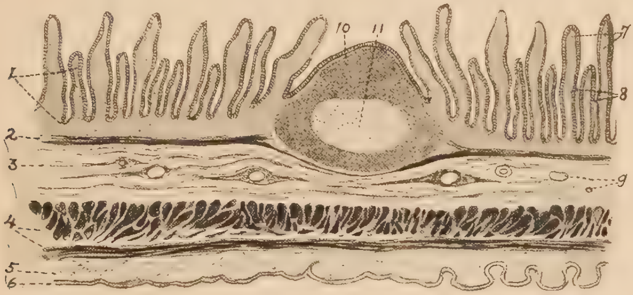

Fig. 6.—Longitudinal cross-section through the wall of the small intestine: 1, Mucous layer; 2, muscularis mucosæ; 3, submucous layer; 4, muscular layer; 5, subserosa; 6, serous layer; 7, intestinal villi; 8, intestinal glands (Lieberkühn); 9, blood-vessels; 10, solitary lymph nodule; 11, center of same.

The serous coat is formed by the visceral layer of the peritoneum. The muscular coat consists of an internal circular layer and an external longitudinal layer, the former being considerably the thicker.

They consist of bundles of unstriped muscular tissue supported by connective-tissue fibers. The submucosa consists of connective tissue in which blood-vessels, lymphatics, and nerves ramify.

The mucous membrane comprises a thin muscular layer (muscularis mucosæ), containing circular and longitudinal fibers and the tunica propria of the mucous membrane, which is made up principally of reticular connective tissue, with leukocytes, glands, villi, and an epithelial covering.

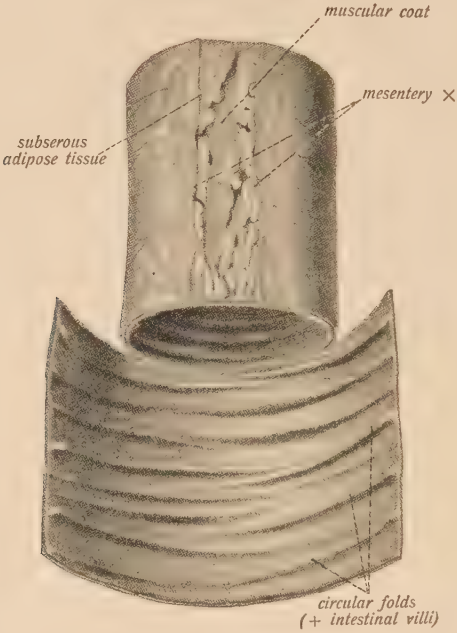

Fig. 7.—A portion of the jejunum showing the circular folds or valvulæ conniventes of Kerkring (Sobotta).

The mucosa is of a grayish-red color, appears velvety, and forms crescentic folds, set transversely to the long axis of the intestine (valvulæ conniventes of Kerkring, Fig. 7). Each valve extends from one-half to two-thirds of the circumference of the gut, and they may be 2 inches long and $\frac{1}{3}$ inch wide. They begin a short distance below the pylorus, at the middle of the jejunum commence to diminish in size, and gradually disappear at the lower part of the ileum. They serve to increase the absorptive surface of the mucous membrane.

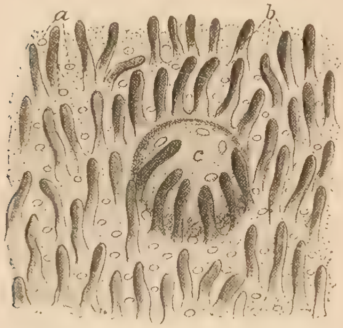

Fig. 8.—Mucous membrane of ileum; *a*, Intestinal glands (Lieberkühn); *b*, intestinal villi; *c*, solitary lymph-nodule (follicle).

**Microscopic Anatomy of the Small Intestine.**—The inner surface of the small intestine is composed of villi and glands, the surface being covered by a layer of columnar epithelial cells, with striated borders and some goblet-cells.

The villi are formed chiefly by elevations of the tunica propria of the mucous membrane. They are from 0.5 to 0.7 mm. in height and about 0.1 to 0.2 mm. in width, and number about ten millions (Fig. 8).

Each villus has a central space for chyle, which cavity is covered with endothelial cells and connects with the lymphatics of the mucosa. The villus has blood-vessels and muscular fibers which are derived from the muscularis mucosæ. It expands when filling up with blood, and when the muscle contracts it shrinks.

The villi thus have an action of suction and pumping, and also form the chief organs for absorption in the small intestine. Around the villi are numerous glands; the tubular glands of Lieberkühn and the acinous glands of Brunner. The latter are confined to the duodenum.

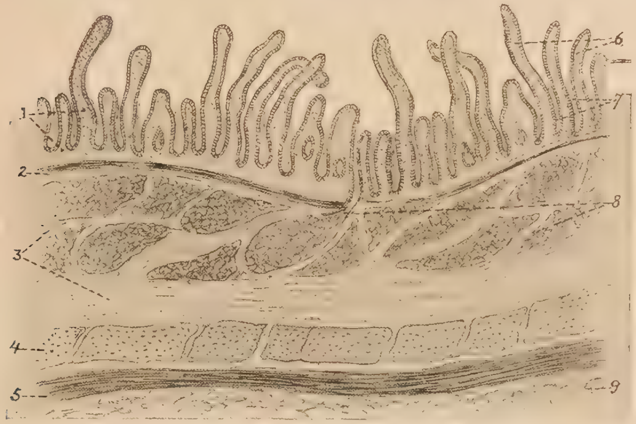

Fig. 9.—Longitudinal cross-section through wall of duodenum: 1, Mucous layer; 2, muscularis mucosæ; 3, submucous layer; 4, circular muscular layer; 5, longitudinal muscular layer; 6, intestinal villi; 7, intestinal glands (Lieberkühn); 8, Brunner's duodenal glands; 9, serous layer.

The glands of Lieberkühn resemble in structure the tubular glands of the stomach, and cover almost the entire surface of the small and large intestine. Each tubule is from 0.3 to 0.4 mm. long, and opens without ramifications. They form the chief organ of intestinal secretion and number over forty millions.

Brunner's glands are found only in the duodenum and are most abundant at its commencement, diminishing in number lower down. They lie beneath the mucosa, being embedded in the submucosa. They resemble the pyloric glands of the stomach, but are more branched and convoluted, and their ducts are longer. They are lined with columnar epithelium. The duct of the gland passes through the muscularis mucosæ and opens on the surface of the mucosa (Fig. 9).

*Solitary follicles* (or glands) are scattered throughout the mucous membrane of the small intestine and are most numerous in the lower ileum. They have a diameter of 2 to 6 mm. (Fig. 10).

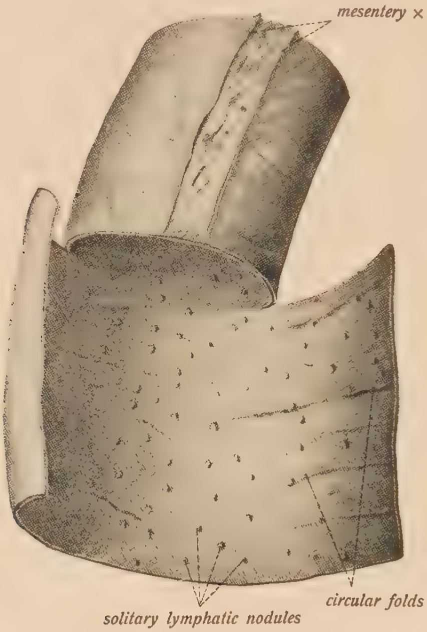

Fig. 10.—A portion of the ileum showing solitary lymphatic nodes (Sobotta).

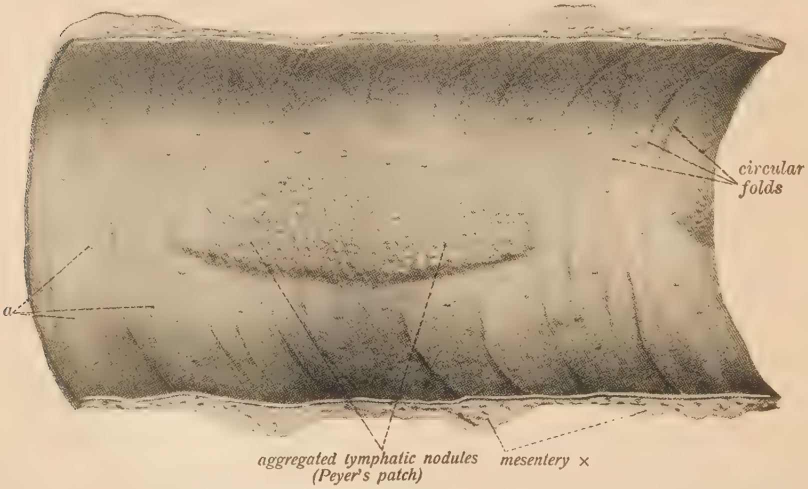

Fig. 11.—A portion of the ileum, cut open along the line of attachment of the mesentery, showing Peyer's patch and solitary lymphatic nodes (*a*) (Sobotta).

The follicles consist of a dense retiform tissue packed with lymph-corpuscles and permeated by capillaries. They have no ducts. The spaces in this tissue are continuous with lymph-spaces at the base of the gland and the base of the follicle is in the submucous tissue. The gland enters the mucous membrane, causing a slight projection of its epithelial layer. Lymph-cells develop in these follicles. There are no villi on their surface.

These follicles are scattered singly through the intestine as solitary glands, or collected into groups, known as Peyer's patches or plaques, or as the agminate glands. These last may be from 1 to 3 inches long and ½ inch wide, usually oval, with the long axis parallel with that of the intestine. They lie generally opposite the attachment of the mesentery, are twenty to thirty in number, and are found chiefly in the ileum, though a few are present in the jejunum (Fig. 11).

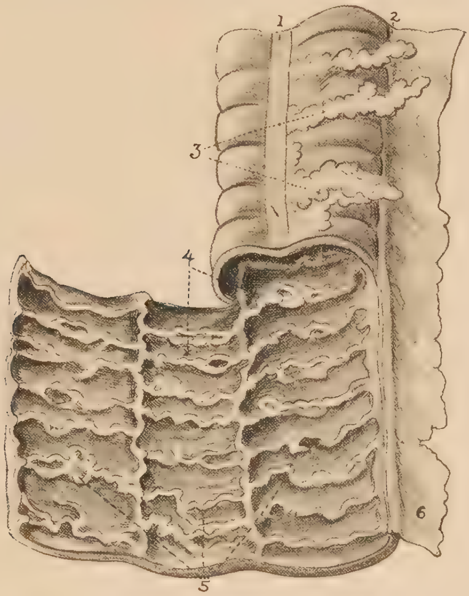

Fig. 12.—Partial section of colon: 1, Free tenia; 2, tenia mesocolica; 3, appendices epiploicæ; 4, mucosa; 5, semilunar folds; 6, mesocolon.

**Anatomy of the Large Intestine.**—The large intestine, which is about 5 to 6 feet (1.5–1.8 meters) long, extends from the termination of the ileum to the anus, and is divided into the cecum (or caput coli), the colon, and the rectum. Its caliber is largest at the cecum, and this gradually decreases until it reaches the ampulla of the rectum, when it again increases in size.

The large intestine, excepting the rectum, is characterized by three longitudinal unstriated muscular bands or teniæ (Fig. 12), with sacculation of the walls between these bands and by the appendices epiploicæ, or external pouches formed by the peritoneal covering and containing fat.

The circular muscular fibers also accumulate in bands with intervals between them, thus forming expansions or semilunar folds across the colon (haustra coli) (Fig. 13).

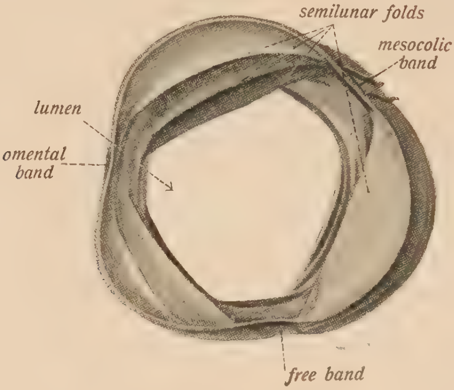

Fig. 13.—A segment of the colon (Sobotta); haustra coli.

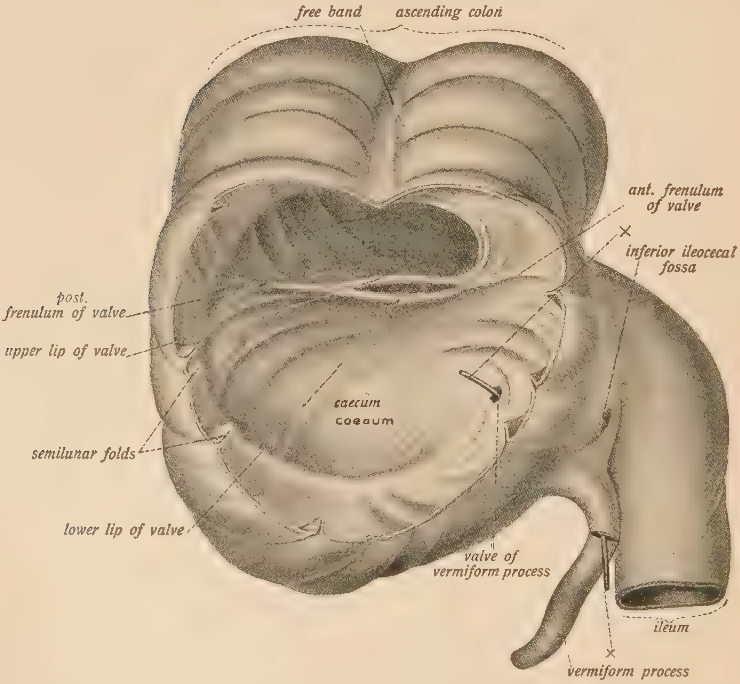

Fig. 14.—The cavity of the cecum (Sobotta).

The cecum (caput coli) is that part of the colon lying below the ileocecal valve (Fig. 14). It is about 3 inches (7.5 cm.) broad and 2½ inches (6.3 cm.) long, and lies in the right iliac fossa above the outer half of Poupart's ligament, being completely covered by peritoneum. When filled, it is situated close to the abdominal wall. The vermiform appendix, a small blind tube, hollow nearly to the tip, is given off generally from the posterior and inner portion of the caput coli, about $\frac{11}{16}$ inch (1.7 cm.) below the ileocecal valve. Its average length is $3\frac{3}{4}$ inches (9.2 cm.). It may be much shorter or longer. The diameter is about $\frac{1}{4}$ inch (6 mm.) at the base and $\frac{3}{16}$ inch (5 mm.) at its apex. It may be slightly larger at the middle. It is usually guarded by a valve (crescentic fold) where it enters the cecum. It has a meso-appendix (mesentery) only extending about two-thirds its length. The position of the appendix is not fixed and it may point in various directions. The colon is depicted in Fig. 21.

The ascending colon, about 8 inches (20 cm.) in length, extends vertically upward from the cecum to the inferior surface of the right lobe of the liver to the right of the gall-bladder, at which point it bends to the left (hepatic flexure). It passes along the posterior abdominal muscles and lower part of the right kidney, and is in relation to the abdominal wall in front. It is bound posteriorly by connective tissue to the muscles, and is only covered by peritoneum anteriorly and laterally.

The transverse colon, with an average length of 20 inches (51 cm.), extends from the hepatic flexure beneath the liver transversely across the abdominal wall, with a slightly downward and forward convexity at its center, to the spleen in the left hypochondrium (splenic flexure). It has a long mesentery, transverse mesocolon, connecting it with the posterior abdominal wall, and is the most movable part of the large intestine. It usually corresponds to a line separating the umbilical and epigastric regions. Formerly it was believed that the normal transverse colon lay just above the umbilicus, but it has been demonstrated by the *x*-rays that a moderate degree of sagging is most frequent and that in most cases it lies below the umbilicus. This occurs frequently without any disturbances and with no ptosis of the stomach. The liver, gall-bladder, greater curvature of the stomach, and lower end of the spleen lie above it; the small intestine lies below; the descending duodenum and small intestine behind; the great omentum and abdominal wall in front.

At the splenic flexure below the lower end of the spleen, the colon turns downward (descending colon). This is about $8\frac{1}{2}$ inches (21.5 cm.) long, and extends from the splenic flexure vertically through the left hypochondriac and lumbar regions to the sigmoid flexure. It is covered anteriorly and laterally by the peritoneum, and passes down in front of the left kidney and quadratus lumborum and iliac muscles, to the left iliac fossa into the sigmoid flexure.

The sigmoid flexure of the colon (S. romanum) is an S-shaped curve, about 13 inches (31 cm.) long, beginning at the iliac crest and ending at the brim of the true pelvis opposite the left sacro-iliac articulation. The upper, or colic, limb tends down, inward, and forward toward Poupart's ligament, while the lower, or rectal, limb hangs down into the true pelvis, where it joins the rectum. The sigmoid flexure has a complete peritoneal covering, or mesentery, is very movable, and is the narrowest portion of the large intestine.

The rectum, which is about 8 to 9 inches (20–23 cm.) long, passes from the left sacro-iliac junction obliquely to the middle of the sacrum and follows it down to the bottom of the pelvis to about 1 inch (2.5 cm.) below the tip of the coccyx, where it passes downward and backward to end in the anus. It is divided into three portions.

Only the first part, 3½ inches (9 cm.), of the rectum is completely invested with peritoneum (mesorectum), and it is attached to the sacral vertebræ. The second part is partially invested with peritoneum (pouch of Douglas), which lies anteriorly and ascends over the bladder or vagina. The third part has no peritoneal investment.

The lower half of the rectum passes between the organs occupying the pelvic floor and is adherent to them by connective tissue. The rectum is surrounded by connective tissue below the pouch of Douglas. It is widest at the point opposite the prostate, there forming the ampulla of the rectum.

The outer longitudinal muscular fibers of the rectum are not arranged in teniæ, as in the colon, but are present in all parts of its circumference. The inner circular layer of muscle-fibers increases in density from above downward and forms a thick ring at the anal opening (the internal sphincter). The walls of the rectum are connected at the anus with the sphincter ani and levator ani muscles, which are of importance in defecation.

**Arterial Supply of the Cecum and Colon.**—They are the ileocolic, colica dextra, and colica media from the superior mesenteric artery; the colica sinistra and sigmoid from the inferior mesenteric artery.

**Veins of the Cecum and Colon.**—These are the superior and inferior mesenteric, emptying into the portal system.

**Lymphatics of the Cecum and Colon.**—Those of the sigmoid colon empty into the lumbar glands; those of the rest of the colon into the mesenteric glands.

**Nerves of the Cecum and Colon.**—The cecum, ascending colon, and right half of the transverse colon are supplied by the superior mesenteric plexus, a branch of the celiac plexus. The rest of the colon, including the sigmoid flexure, is supplied by the inferior mesenteric plexus, a branch of the aortic plexus.

**Arterial Supply of the Rectum.**—They are the superior hemorrhoidal (of the inferior mesenteric); the middle hemorrhoidal (of the internal iliac); the inferior hemorrhoidal (of the internal pudic); branches from the sacromedia (of the abdominal aorta); branches from the sciatic (of the internal iliac); in the female, branches from the vaginal.

**Veins of the Rectum.**—They are chiefly from the superior hemorrhoidal, passing to the inferior mesenteric and to the portal system; part of the other hemorrhoidal veins empty into the internal iliac veins, and so enter the general venous system.

Anastomosis thus occurs in the rectum between the portal and the general venous system, and there is a communication also with the other abdominal veins.

**Lymphatics of the Rectum.**—They empty into the sacral and lumbar glands from the rectum; from the anus, into the inguinal glands.

**Nerves of the Rectum.**—The cerebrospinal nerves come from the sacral plexus; the sympathetic nerves from the inferior mesenteric and superior hypogastric plexus.

## HISTOLOGY OF THE LARGE INTESTINE

The large intestine (colon), like the small intestine, consists of four coats: serous, muscular, submucous, and mucous.

The longitudinal muscular fibers of the large intestine, however, are arranged in three bands (teniæ) running along the wall, as already described.

The serosa and submucosa resemble those of the small intestine in structure.

The mucosa of the large intestine differs from that of the small intestine in that the villi and circular folds of Kerkring (valvulæ conniventes) are absent. The glands of Lieberkühn are somewhat longer and at times curved.

The mucous membrane of the rectum is thicker, redder, and more generously supplied with blood-vessels than that of the colon.

When the rectum is empty, the mucous membrane of the upper part is thrown into a multitude of superficial transverse velvety folds. From two to seven folds (Houston's valves) are made more prominent by distention (Gant).

Just above the anus are a number of longitudinal folds (columns of Morgagni), extending for $\frac{1}{3}$ to $\frac{3}{5}$ inch (8.46–15.23 mm.).

The mucous membrane of the rectum consists of columnar epithelium, except at the lowest portion, a narrow layer of stratified pavement-like epithelium, transitional between skin and rectal mucosa. The upper part of the rectum resembles the colon.

## CHAPTER II

# PHYSIOLOGY OF DIGESTION

The stomach and intestines form an important part of the digestive tract, and in order to understand their functions it will be necessary to review briefly the process of digestion. This term includes those processes which convert the food into such condition that it becomes fit to enter the circulation and afford nutrition to the human organism. These changes are brought about by means of certain ferments contained in the saliva, gastric juice, bile, pancreatic juice and intestinal juice, which are a part of the human organism at birth.

There are certain fermentative and putrefactive processes which take place in the gastro-intestinal canal, the result of bacterial invasion, which play an important part in the physiology and pathology of this tract.

The first ferment (ptyalin) with which the food comes in contact by the act of chewing, is found in the saliva. The latter is alkaline in reaction, of low specific gravity (1.002 to 1.0009), and contains water, ptyalin, mucus, epithelia, albumin, and salts.

The ptyalin, which converts starch into maltose, or sugar, begins its action on the food already in the mouth, but the principal work is done during the first period of digestion within the stomach. The food passes into the fundus and accumulates there to form a mass, and it is not until twenty to thirty minutes later that the first movements of the stomach begin to appear. Under normal conditions during this early stage of gastric digestion the free hydrochloric acid is becoming combined acid, and the action of the ptyalin continues; but if the free hydrochloric acid be excessively secreted, further digestion of the starch is interfered with; this physiologic fact has a bearing on the treatment of hyperchlorhydria. We must remember that thorough mastication of the food, which promotes salivary secretion, and the care of the teeth have an important bearing on digestion. It has been demonstrated that acid fermentation in the mouth interferes with the action of the saliva, and that cleansing the mouths of nursing infants will diminish fermentative processes in the gastro-intestinal tract.

Hemmeter has apparently recently demonstrated on dogs that the salivary glands secrete a chemical substance (a *hormone*) during mastication which *passes into the blood* and starts up the secretion of the gastric juice. After extirpation of the glands, HCl and rennin were much reduced. Intravenous injection of salivary gland extracts partially restored gastric secretion.

## THE GASTRIC JUICE

Hydrochloric acid was discovered in the gastric juice by Prout in 1824; and Beaumont, in 1833, by his experiments on St. Martin with his gastric fistula, greatly advanced our knowledge. Schwam, in 1836, discovered the pepsin ferment; and Bedder and Schmidt, in 1854, showed that the acid of the gastric juice is hydrochloric acid.

The gastric juice is a clear, colorless fluid, of an acid reaction and a specific gravity of 1.002 to 1.003. It contains water, salts, inorganic matter, proteins, hydrochloric acid, pepsin, rennet, and recently a fat-splitting ferment has been discovered (lipase).

The quantity of this secretion in twenty-four hours is estimated to be about 3 pints. The degree of acidity varies from 0.1 to 0.2 per cent. Pepsin and rennet when first secreted are inactive bodies, known as pepsinogen and rennet-zymogen, but on coming into contact with the hydrochloric acid become converted into active pepsin and rennet. Various theories have been advanced to explain the production of the gastric juice, how an inorganic acid comes to be secreted by the blood which is of alkaline reaction. We are so far forced to accept the view that the secretion of the gastric juice must be due to the specific action of the cells.

Through the combined action of hydrochloric acid and pepsin, it converts the albuminates into propeptones and peptones, which are more soluble. The rennet ferment curdles the milk. A small percentage of fat is split into fatty acids.

The gastric juice has the property of converting cane-sugar into grape-sugar, and gelatin into a soluble peptone which does not coagulate.

Some of the substances contained in the liquefied chyme are absorbed through the stomach-wall, such as peptone, sugar, salts, and possibly propeptone. Meltzer has demonstrated that only a small quantity of water is absorbed in the stomach. The residue of the gastric contents passes into the small intestine, where further digestive processes occur through the action of the other ferments and the principal absorption takes place.

(1) **Secretion of the Gastric Juice.**—Pawlow[1] has discovered many new facts relating to the physiology of gastric secretion. Among these are the following: The amount of gastric juice secreted is proportionate to the quantity of food ingested; the secretion is acid, and the more rapidly it appears, the greater is the quantity of acids produced. The gastric juice appearing *after a bread diet* shows the *most marked digestive activity;* following this is the gastric juice after a meat diet, and the least digestive activity is found after a

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[1] The Digestive Glands.

milk diet. Moreover, every *variety* of food stimulates *a definite quantity of secretion.*

*Quantity of Secretion.*—The average quantity of juice secreted every hour is one and a half *times less after a bread* diet than after a milk or meat diet, the digestion consuming a longer period of time. A latent period occurs between the ingestion of food and the appearance of the secretion, lasting from four and a half to ten minutes. *Nervous influences* play an important rôle in the secretion of the gastric juice as is demonstrated by the fact that after feeding a dog in whom an esophagotomy has been performed (sham feeding), a certain quantity of gastric juice will appear in the gastric fistula within a few minutes. The sensations of taste, smell, sight, and the presence of food within the mouth reflexly stimulate the secretory nerves in the vagus. Sham feeding has no longer any influence on the secretion of gastric juice if the vagus has been severed, demonstrating that this is the secretory nerve of the gastric glands. As already mentioned, the sight of food will cause secretion of the gastric juice (appetite juice), since the appetite is a strong stimulant to the secretory nerves. Hertz and Sterling, however, in studying the psychic gastric juice in a gastrostomy patient,[1] found that in this case there was not the intimate correlation in the simultaneous presence of appetite and a flow of gastric juice, nor did mastication of sapid substances cause the flow of an "appetite juice." They refused, however, to generalize from this single subject. Pawlow, moreover, found that *further secretion* of the gastric juice is due to *certain substances* contained in the foods, known as *secretogogues* (which stimulate further secretion). Pure mechanical stimulation is not the cause. *Meat juice* and *beef extracts* are marked *stimulants* to the flow of gastric juice, while milk is less so, and water is a weak stimulant. Pawlow further claims that the ash of meat, sodium chlorid, hydrochloric acid, and soda do not stimulate the secretion, and that egg-albumen and pure peptone have no effect. Starch, cane-sugar, and grape-sugar have no influence, while starch paste and meat extract together markedly increase this secretion. Fats have no influence on the secretion of gastric juice, and when introduced with other food diminish this secretion. Edkins[2] claims that the secretogogues, whether they are preformed in the food, or formed from it in the process of digestion, act upon the pyloric mucous membrane and form *gastrin* (or gastric secretin), which is absorbed into the blood and then carried to the gastric glands to stimulate their activity. Starling shows that this is similar in action to the secretin in producing pancreatic secretion. He designates these internal secretions as "hormones."

(2) **The Motor Function of the Stomach.**—The *motor function of the stomach* is of importance in the process of digestion, as by its active

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[1] Deutsche med. Woch., Aug. 4, 1910.

[2] Journal of Physiology, 1906, xxxiv, p. 133.

and passive movements physical changes are brought about in the ingesta. The movements transmitted by the diaphragm and abdominal muscles assist the action. The food is brought more closely in contact with the stomach-wall and becomes more liquefied. The pylorus opens and closes at intervals, and allows the entrance of the chyme into the small intestine, at an interval of about twenty seconds, according to Cannon, depending on the acidity of the chyme and the consistency of the food. About two to three hours after a small meal, and six to seven hours after a large meal, the stomach is empty.

Cannon[1] has studied the movements of the stomach by the *x*-rays, by administering food mixed with bismuth, and by observation with a fluoroscope. He found that the contractions start in the middle of the stomach and pass toward the pylorus at regular intervals. The pyloric end of the stomach lengthens out and the peristaltic waves increase during advanced digestion. At intervals the pylorus relaxes and the contraction squeezes part of the chyme into the duodenum.

He[2] found that carbohydrates pass out the soonest after ingestion and require only about one-half the time that proteins do for gastric digestion. If the protein is given first, the passage of the carbohydrate will be delayed. Fat when taken alone remains for a considerable time in the stomach, and if combined with other food, the exit of the latter is delayed. It is believed that chemic stimuli, such as hydrochloric acid, control the opening and closing of the pylorus. With achylia gastrica, however, the motor function of the stomach, in spite of the absence of hydrochloric acid, is frequently normal. The consistency of the chyme, to which Cannon refers, and possibly some new formed chemical constituents, may be responsible for the regularity of the pyloric reflex in this case. Bassler[3] has made some rather interesting observations with the *x*-ray in this regard.

Grützner and Cannon[4] have demonstrated, moreover, that the waves in the fundus are very slight and do not affect the contents in that portion of the stomach for a considerable period of time, so that the food lying there does not immediately mix with the acid gastric juice. This is an important fact in connection with the salivary digestion of the starches, so that ptyalin digestion may in some cases continue for an hour, before it is inhibited by the acid gastric juice. The swallowed food arranges itself in consecutive layers in the stomach. Physiologic observations apparently show that nature provides against the danger of diluting the gastric contents during digestion by the drinking of fluids during the meal; since O. Cohnheim observes that even in a stomach containing considerable food, the ingestion of a large quantity of fluid causes a separation of the food from the lesser curvature, along which the fluid passes rapidly into the duodenum.

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[1] American Journal of Physiology, 1898.

[2] Ibid., 1904.

[3] Diseases of the Stomach and Upper Alimentary Tract.

[4] Archiv. f. die gesammte Physiologie, p. 106, 463.

F. Hoffmeister, Schuetz, Rieder, Brunner, and Groedel[1] describe several phases in the motor mechanism of the stomach:

*Phase 1.*—This occurs in the fundus. There are rapid undulatory movements, slightly progressing, also oscillations of contraction around the gastric contents (peristalsis), which maintain, at the same time, a certain tonus of the wall. At the lowest point of the great curvature the contractions deepen, and about four fingers from the pylorus there is a constriction which gradually deepens and, meeting a similar constriction of the lesser curvature, forms a temporary occlusion of the lumen at this point—the sphincter antri pylori. In the fundus, the shaking apparatus, the chemical impregnation of the food takes place and a sorting of the chyme by the sphincter of the antrum.

*Phase 2.*—This occurs within the antrum, the so-called accessory stomach. The contractions are of longer duration and tonic in character. There is also a shortening in the longitudinal direction. Thus the fluid food which cannot escape through the sphincter antri is pushed through the open pylorus. The antrum also squashes the contents.

*Phase 3.*—The contraction of the antrum subsides, the pylorus closes, and the food which has not escaped through it into the duodenum floats back through the relaxed sphincter antri into the fundus. When the chyme is thick, this antiperistaltic action may be quite vigorous. It is the only antiperistaltic action in the normal stomach. It is now believed that the so-called sphincter antri is merely the point of maximum peristaltic contraction.

If atony of the stomach be present, motor insufficiency and stasis result, and the latter favors fermentation and putrefaction. It has been demonstrated that the presence of hydrochloric acid in considerable quantities does not prevent these conditions if stasis be present.

## INTESTINAL DIGESTION

Under normal conditions, when the chyme enters the duodenum its reaction is acid. It is here subjected to the influence of the bile, pancreatic juice, and intestinal secretions, all of which, in their action, have a more or less close interdependence upon each other.

**Bile.**—The bile in the intestine precipitates the pepsin from the chyme. This, however, is soon dissolved. Others hold that albuminoids are precipitated from the chyme, together with the pepsin, and are then more readily absorbed. It does not have a deleterious effect on the pancreatic digestion. Bile contains a weak amylolytic ferment.

The bile is one of the chief factors in gradually *altering the reaction of the chyme to neutral or alkaline*, and *it strongly supplements the*

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[1] Archiv. für exper. Pathologie, xx.

*action of the pancreatic juice in emulsifying fats, and its absence or diminution lessens this function.*

Pawlow believes that it may augment slightly the effects of the other pancreatic ferments.

One of its chief functions *is undoubtedly excretory,* and through it many of the useless products of metabolism are eliminated. When absorbed into the system it acts as a poison and produces a definite toxemia.

The bile is a clear, tenacious mucoid fluid with an alkaline reaction, consisting of water, bile-acid salts (glycocholic and taurocholic acid), pigments (bilirubin and biliverdin), mucin, cholesterin, lecithin, soaps, fats, etc.; about 500 to 600 c.c. are excreted in twenty-four hours.

Wm. H. Porter[1] believes that the precipitation of the pepsin from the chyme *through the action of the bile* is necessary for the action of the pancreatic ferments, and the enterokinase of the intestinal juice, as the pepsin, as long as it remains active, inhibits their action.

Some consider that it has antifermentative and antiputrefactive powers, and that it helps to maintain the nutrition of the epithelial cells. Lindenberger has shown experimentally that a small amount of bile combined with .05 per cent. of lactic acid prevents putrefaction in an infusion of pancreas, while lactic acid alone has no effect.

Von Noorden holds that bile has no antiseptic action.

Clinically, we at times see cases of indicanuria with the presence of bile in the urine, with light-colored stools in which there is no *permanent disappearance of indican,* though there is a temporary improvement after the use of calomel, etc., until the biliary excretion into the intestine is restored to normal. The internal use of the bile-salts, which can be given as inspissated bile, seems to be of some value. Bile possesses slightly purgative qualities.

The liver has special functions. Many of the substances taken up by the digestive process are stored there until used in the system, namely, some of the peptones and sugar in the form of glycogen. It excludes some poisonous matters from the circulation. Thus it is the chief organ for the removal of indol and poison, such as curare or of various autotoxins. Urea is also formed in the liver.

**Pancreatic Juice.**—The pancreatic juice is the principal factor of digestion in the intestinal canal. It is clear, colorless, alkaline, sticky and odorless, quite albuminous, containing water, solids, proteins, and inorganic matter.

The most important constituents are the three ferments:

*Amylopsin,* an amylolytic enzyme, which converts starch into dextrin and maltose, and still further into glucose. Cane-sugar is converted into grape-sugar, while milk-sugar is unchanged. Some of the cellulose ferments form marsh-gas and various acids. The activity of pancreatic diastase is increased by very small quantities of acids (Chittenden).

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[1] Indicanuria a Danger Signal, Postgraduate, 1907.

*Steapsin*, a lipolytic ferment. This acts upon fats by splitting them into fatty acids and glycerin; and this action is increased by the bile. The fatty acids combine with the alkalis in the intestines to form soaps, which aid in the emulsifying of fats, and thus promote their absorption. The emulsification occurs in an alkaline medium, or in contact with the alkaline secretion of the mucous membrane.

*Trypsin*, a proteolytic ferment, changes the proteins into albumoses and peptones. There is probably a rennet-zymogen also.

The ferment acts in a neutral or alkaline medium, though slight degrees of acidity seem to favor it. Indol is a product of intestinal putrefaction, and not from the action of the pancreatic ferment, as was formerly supposed. The same is true of hypoxanthin. Some hold there is a lab ferment and a ferment "nuclease." (See Section on the Pancreas.) There is an internal secretion of the pancreas influencing the metabolism of sugar.

The secretion of the pancreatic juice is not reflex, but is due to direct excitation of the cells of the pancreas by secretin (Starling). This is formed from "prosecretin" in the mucous membrane of the *duodenum* and *jejunum* and reaches the pancreas by the blood-stream. The passage of the hydrochloric acid in the chyme over these portions of the small intestine stimulates the production of secretin or, rather, splits it off from the prosecretin, which is present in the mucous membrane.

The **intestinal juice** (**succus entericus**) consists of water, albumin, mucin, and salts. It seems to neutralize the acids formed by the fermentation of the carbohydrates, and the presence of mucin shows it to be of service in aiding peristalsis.

Pawlow's experiments demonstrate that it augments the activity of the pancreatic ferments, especially of trypsin. The fat-splitting and amylolytic pancreatic ferments are augmented by the succus entericus from all parts of the small intestine, while that from the duodenum augments chiefly the proteolytic ferment. Ptyalin and several enzymes have been found in the intestinal juice.

The intestinal juice contains three enzymes acting on the carbohydrates: Maltose, which acts on maltose; invertin, which acts on cane-sugar; and lactose, which acts on milk-sugar.

Pawlow found enterokinase, a ferment of other ferments, which is believed to be necessary to excite intestinal ferments into activity. It probably acts chiefly in *converting trypsinogen into trypsin*.

Erepsin, another ferment, is also present, which transforms hemialbumose into other bodies, completing the work of the pepsin and trypsin.

**Organized Ferments.**—There exist in the intestines fermentative and putrefactive changes produced by micro-organisms.[1] At birth the gastro-intestinal tract is sterile, but rapidly, by the ingestion of food and through the air and by the anus, bacteria of various types enter this tract.

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[1] Proceedings American Medico-Psych. Association, April, 21, 1905. Some Observations on the Relations of the Gastro-intestinal Tract to Nervous and Mental Diseases (Kemp).

Herter[1] has estimated their number at one hundred and twenty-six billions for the daily human excreta. Many of them are no longer living. He considers the chief function of the obligate bacteria (Bacillus lactis aërogenes, Bacillus coli, and Bacillus bifidus) to be their capacity for checking the development of other types of organisms capable of doing injury, though *they themselves under certain conditions may produce much harm.* Many other varieties are described. Some observers believe that there is a so-called normal fermentative process which aids in the digestion of cellulose, though Bergman claims there are enzymes (intracellular) which decompose it.

The fermentative processes in the small intestine caused by the action of bacteria on the carbohydrates (Bacillus lactis aërogenes) leads to the formation of ethyl alcohol and various organic acids, such as lactic, acetic, paralactic, succinic, biliary acids and albumin, peptone, mucin, sugar, etc. These organic acids are believed to prevent putrefaction within the intestines, to partly check the decomposition of the carbohydrates, and to aid in producing intestinal peristalsis, which render putrefaction less likely. The lactic acid in koumyss, matzoon, bacillac and lactone buttermilk, and even in plain milk (to a slighter degree), is believed to lessen putrefaction. In the lower jejunum and ileum the reaction is acid.

When the intestinal contents pass into the colon the reaction becomes alkaline, fermentation stops, putrefaction begins, and the fecal odor appears. The colon bacilli are marked factors in this process. The decomposition of the albuminates caused by bacteria goes much further than that produced by pancreatic digestion. Albumoses, peptones, lysin, ammonia, amido-acids, etc., are produced in both cases, but with putrefaction the process goes further and we have new products formed, such as indol, skatol, paracresol, phenol, various acids and gases, such as sulphuretted hydrogen, marsh-gas, carbon dioxid, etc.

Some of these products of decomposition are eliminated unchanged in the urine, such as the oxyacids, others, like the phenols, after further oxidation; others, like indol and skatol, after combination with ethereal sulphuric acids. For example, indol forms an indoxyl-potassium sulphate or indican, and is so eliminated in the urine; and thus may be an indication of the amount of putrefaction occurring in the intestines.

The causes of indicanuria are various, such as excessive protein diet, catarrh of the small intestine causing alterations in the mucosa and increased intestinal putrefaction therefrom, typhoid, cholera, a pus-cavity, constipation, alimentary putrefaction, decrease of normal digestive fluids, intestinal obstruction, and peritonitis.

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[1] Bacterial Infections of the Digestive Tract.

Certain drugs, such as salol, salophen, and creosote, will give nearly similar reaction, while urotropin will cause its disappearance. These possible conditions must all be considered.

As the intestinal contents pass through the large intestine they become thickened through the absorption of fluids and are at last eliminated as feces. These comprise the remains of undigested material, excretory material from the intestines, and many microorganisms.

The quantity of feces depends upon the character of the food, being greater after a vegetable diet. The average amount after a mixed diet is about 100 to 150 gm.

The reaction of the feces is ordinarily alkaline, though occasionally variable. The odor is chiefly due to skatol, and the color is a light or dark brown.

**Absorption from the Intestines.**—Absorption chiefly occurs in the small intestine.

The *proteins* are usually changed into albumoses and peptones before absorption occurs. Albumin may be absorbed as such, though not as quickly.

Absorption of the albumoses and peptones takes place by the capillaries and not by the lacteals. They are reconverted into albumin before reaching the blood-current, probably either by the epithelial cells or leukocytes. It has been shown experimentally that they do not reach the blood as peptones.

Albuminates from animal food are more completely absorbed than those from vegetable food, on account of the indigestibility of the cellulose and the increased peristalsis caused by the latter.

**Absorption of the Carbohydrates.**—These are chiefly absorbed as monosaccharids through the capillaries of the villi, enter the liver through the portal vein, and are retained as glycogen for use in the animal economy.

If sugar is absorbed in excess, it may enter the general circulation and be excreted by the kidneys, so-called alimentary glycosuria. It may also cause diarrhea.

Carbohydrates, as starch, are absorbed without difficulty.

Glucose, levulose, and galactose are absorbed as such, while cane-sugar and maltose are first changed to these products. Milk-sugar is unchanged and absorbed as such, or undergoes lactic-acid fermentation.

**Absorption of Fats.**—The greatest amount is absorbed as an emulsion (both fats and fatty acids), though some absorption takes place in the form of soaps. They become neutral fats after absorption. They enter the lacteals probably through the action of the epithelial cells of the intestinal wall and reach the thoracic duct.

The absorptive power for fat in the small intestine is considerable, probably over 300 gm. per day. Olive oil and butter (fats with a low melting-point) are absorbed more quickly than mutton fat, for instance (fat with a high melting-point), and a free fat, such as butter, is taken up more quickly than bacon, which contains considerable connective tissue.

Water, salts, some of the secretory juices, and bile are readily absorbed.

Disease or removal of the pancreas stops the absorption of fats, except of milk, of which part is absorbed in emulsified form.

**Absorption in the Large Intestine.**—Water, fluids, and salts are well absorbed, in fact, markedly absorbed, as is noted by the change in the character of the intestinal contents. Albumin and carbohydrates are absorbed in considerable amount and fats in small quantities. Advantage is taken of this fact for the employment of nutritive enemata.

**Intestinal Peristalsis (Motor Function).**—The contents are thoroughly mixed by the movement of the intestines, and the residuum left after digestion is expelled through the anus.

There are four types described:

*First.*—Cannon has shown that the small intestine is much more actively engaged in the segregation or segmentation of food than in its mere propulsion. One type of motility is given over to the segmentation of food in short blocks in the small intestine, thus giving sufficient time for thorough digestion as well as absorption. During this process the mucous membrane, with its large absorbing valvulæ conniventes, is thrust into the food mass, thus exposing its maximum surface to absorption.

*Second.*—The second movement is peristaltic, and the intestines contract at a certain point and then relax, and continue this in successive segments or blocks progressively toward the anus, pushing the contents forward (*ordinary peristaltic movements*).

*Third.—Oscillating movements*, by which the coil is moved to and fro along the mesentery, with no particular contraction. The contents are mixed up by these movements and not propelled forward.

*Fourth.—Rotary movements*, by which a coil contracts in a circular direction rapidly along the intestines for 15 or 20 cm. in a violent manner.

The last is usually pathologic and occurs when there is considerable gas, after indiscretions in diet, or with stenosis. It is observed only in the small intestine.

Peristaltic action is much more rapid in the small intestine. After ingestion of a small meal the stomach becomes empty in about two hours, the small intestine in the same length of time; but in the large intestine it takes at least twenty hours before the contents are expelled.

Nothnagel has never seen a physiologic antiperistalsis (reversed peristalsis) beginning from the anal direction upward toward the stomach, though some[1] describe an intermittent antiperistalsis occurring in the ascending colon. It is said that strong injections of salt water into the colon will produce this effect.

**Nervous Control of Peristalsis.**—Auerbach's and Meissner's plexuses are probably the automatic centers for peristalsis, but there are central agencies. For example, fright or excitement may cause diarrhea.

The splanchnic nerve contains inhibitory fibers for the control of intestinal peristalsis.

Ehrmann claims that the longitudinal muscles are stimulated by the splanchnics and inhibited by the vagus, and the circular muscles stimulated by the vagus and inhibited by the splanchnics.

The chyme acts as the normal stimulus to peristalsis through the nerves. Toxic material that has been ingested or developed in the intestinal canal, indigestible food, organic acids from excessive fermentation, and too hot or too cold drinks, may overstimulate the peristaltic action and be the cause of diarrhea.

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[1] W. B. Cannon, Amer. Jour. Physiol., vol. vi, p. 253, has demonstrated by the Röntgen rays that antiperistaltic movements occur normally in the cecum, ascending and transverse colon, thoroughly churning and mixing the food, and bringing it in more perfect contact with the absorbing surface of the colon.

## CHAPTER III

# INTERROGATION OF THE PATIENT (HISTORY)

In every case suffering from symptoms pointing to the gastro-intestinal tract, before the physical examination is undertaken the patient should be carefully interrogated as to his general history and the past and present symptoms.

I shall briefly indicate the form of taking and preserving the history of such cases:

Date................ Diagnosis............ No. of patient..............
Name............................ Nativity..........................
Age............................. Occupation........................
Sex..............................

*Family History*

Tuberculosis:
Syphilis:
Diphtheria:
Scarlatina:
Measles:
Typhoid:
Gout:

Rheumatism:
Malaria:
Influenza:
Nephritis:
Heart disease:
Disease of liver:

*Habits*

Tea—cups:
Coffee—cups:
Tobacco:
Alcohol:

Sexual excess:
Mastication:
Character of food:

*Past History of Present Complaint*

Began:
Duration:

*Onset*—
- Sudden:
- Gradual:

*Probable cause:*

*Symptoms*—
- Progressed:
- Same in character:
- Changed in character:

*Loss of flesh*—
- Present:
- Increasing:
- Absent:

*Bowels*—
- Constipation:
- Diarrhea:
- Alternating:
- Regular:

- Mucus:
- Blood:
- Odor:

### *Present Condition and History*

*Headache—*
Character:
Location:
Time of:

*Vertigo—*
Nervousness:
Drowsiness:
Sleeplessness:

*Appetite—*
Good:
Anorexia (loss):
Time:
Parorexia (perversion):
Bulimia (canine hunger):
Polyphagia:
Akoria:

*Thirst:*

*Taste—*
Normal:
Bitter:
Time:
Sour:
Sticky:

*Deglutition:*

*Dysphagia—*
With solids:
With liquids:

*Abnormal sensations—*
Bloating:
Fulness:
Time:
Pressure:
Weight:

*Belching—*
Quantity:
Time of:
On full stomach:
On empty stomach:
Odor:

*Regurgitation—*
Water-brash:
Sour:
Time:
*Rumination:*

*Pyrosis (heart-burn)—*
Time:
Duration:

*Pains—*
Location:
Cardialgia:
Gastralgia:
Character:
Time of appearance:
Duration:
Affected by position:
Affected by food or drink:
Circumscribed:
Diffuse:
Radiating:
Sudden:
Gradual:
Relieved by pressure:
Increased by pressure.

*Local tenderness—*
Position:

*Nausea—*
Time:
Affected by food:

*Vomiting—*

Time:
Frequency:
Quantity:
Character of:
Odor:
Taste:
Blood and its character:
Bile:
Mucus:
Easy:
Difficult:
Relief of pain by:

*Stools—*

Regular:
Constipation:
Diarrhea:
Alternating constipation and diarrhea:
Number of movements:
Time of appearance:
Undigested food:
Mucus:
Blood:
General character:
Bile:

*General health and strength—*

Loss of weight:

*Symptoms Referable to Circulatory System.*

*Symptoms Referable to Nervous System*

*Chief Complaint*

After the physical examination has been made, the results should be incorporated with the history, as should also the data secured from examination of the gastric contents and stool.

*General Physical Examination Comprises—*

*Tongue:* *Uvula:* *Tonsils:* *Pharynx:*
*Teeth:* *Esophagus, if history points to the same:*
*Eyes*—as to difficulty in reading or headache therefrom; exophthalmos.
*Nose*—as to nasal discharge.
*Ears*—as to deafness.
*Heart:*
*Lungs:*
*Liver:*

*Stomach—*

Position:
  Normal:
  Dilated:
  Gastroptosis:
Tenderness:
Motor function:
Tympanites:
Gastric analysis:
Tumor:

*Spleen:*

*Kidneys—*

Position:
Urine:

*Intestines—*

Position:
Tender points:
Thickening:
Tympanites:
Borborygmi:

*Rectum—*

Local examination:
In some cases microscopy and fermentation test of stool.

*Nervous system:*
*Weight of patient:*
*Examination of genital organs*—in some cases.

## CHAPTER IV

# GENERAL METHODS OF PHYSICAL EXAMINATION

As patients who complain of digestive disturbances may suffer from disease of other organs which may be the cause of the symptoms, a thorough physical examination should be made in every case.

The first step should be an examination of the heart and lungs. The character and rapidity of the pulse and respiration should be noted, and a specimen of urine requested for analysis.

In all *acutely* commencing processes pointing to *the digestive tract, the temperature should be taken.*

### GENERAL INSPECTION

The general appearance of the patient may afford valuable information. With cancer there is often the sallow and emaciated appearance (cachexia) with anemia; with gastric neurosis the patient may often appear rosy and well nourished; while with ulcer there is frequently marked anemia and the face may have the appearance of suffering. Protrusion of the eyeballs, with inability of complete closure of the lids, taken in connection with tachycardia, are suggestive of Graves' disease, even though no thyroid enlargement be present. The skin should be inspected for eruptions and the exanthemata.

**Oral Cavity.**—This should be carefully inspected. Defective and carious teeth or inflammation or abscess of the gums may give rise to gastric disorders. Disease of the posterior nares or middle ear, with resulting discharges passing into the pharynx, may be factors.

The tongue was formerly regarded as a mirror of the stomach, but it can hardly be so considered, as there are some gastric affections in which the appearance of the tongue is normal; while in smokers, for example, the tongue may be coated and yet no gastro-intestinal disturbance be present. A thick gray or grayish-yellow, moist, coated tongue showing indentations is suggestive of chronic gastritis; while with ulcer it may be dry and red, with a white median stripe, or smooth and moist or slightly furred.

The condition of the mouth, smoking, drinking, and the teeth, have a decided bearing on its appearance. This is also true in reference to the odor of the breath. I hardly deem the tongue diagnostic in diseases of the gastro-intestinal tract, except in association with other symptoms.

With the typhoid state, we have the narrow tongue, with the deep median fissure, thickly furred, the tip and edges being red and denuded, or the dry, brown fissured, and tremulous tongue; while with scarlet fever and in some other acute specific infections there is the so-called strawberry or raspberry tongue, with bright red projecting papillæ.

The uvula is sometimes elongated, and may cause reflex digestive disturbances. In the pharynx and tonsils there may be inflammatory conditions of an acute or chronic type which influence the case.

Inspection of the neck is important. Enlarged thyroid with tachycardia is diagnostic of Graves' disease, and a swelling to the left of the larynx, which increases in size after the ingestion of food, is suggestive of a diverticulum of the esophagus.

**Examination of the Esophagus and Esophageal Diseases.**—The cardinal symptom of esophageal disease is dysphagia, with or without regurgitation. The object of examination is to determine whether a stricture or a diverticulum is present.

Palpation of the esophagus is possible in the neck, usually on the left side behind the trachea. A tumor found here may be a diverticulum distended with food or fluid. It is sometimes possible to empty it by the exertion of pressure. After the administration of considerable water, on rising a *splashing sound* can often be determined by grasping the larynx and shaking the patient's neck. Respiration and swallowing should be avoided during the test.

A brawny swelling, in some cases with subcutaneous emphysema, may result from perforation or rupture of the esophagus, with inflammation, which proceeds to suppuration. An abscess in this locality may be due to caries of the vertebræ.

Auscultation of the esophagus is often of service. Place the stethoscope posteriorly to the left of the spine, at the level of the sixth dorsal vertebra, and at a signal let the patient swallow a mouthful of water. At the instant of swallowing the deglutitory sound is heard, followed in six or seven seconds by the esophageal bruit, which resembles the sound one hears when swallowing saliva. Three to five seconds later there is a second sound, caused by the fluid entering the stomach or by regurgitation of air. If the first sound is delayed longer than seven seconds, or replaced by a splashing or gurgling noise, or if the second sound is delayed longer than five to twelve seconds, partial stenosis may be suspected. If both sounds are absent, there is probably nearly complete or complete stenosis.

Gurgling sounds lasting several minutes and heard along the left side of the spine are probably due to contractions in a diverticulum, or in the dilated portion of the canal above a stricture.

Instrumental examination of the esophagus is made by flexible stomach-tubes of various sizes,—the safest method,—or by flexible solid bougies or sounds.

A practical method of differential diagnosis between stenosis and diverticulum of the esophagus has been devised by H. S. Plummer[1] and successfully employed by C. H. Mayo. The patient is directed at bedtime to swallow three yards of buttonhole silk twist with the assistance of drinking water, and the next morning to swallow three more yards of the continuous thread, if there is an opening through the stricture or diverticulum, the thread will be washed into the stomach, and from there into the bowel, a sufficient distance to stand traction without being readily withdrawn. A whalebone stem with several sizes of olive tips is employed for the examination. There is an opening at the side of each tip, the channel emerging at its apex. The thread which is employed as a guide passes through the tip of the olive and out at its side. The instrument is passed down the esophagus on the thread, which is held loosely until an obstruction is encountered. If this is due to a stricture, the tip *will not change its level* when the thread is tightened; but if there is a diverticulum, the probe will be elevated to the level of the opening into the lower esophagus (Figs. 15 and 16). This proves at once the existence of a pocket and

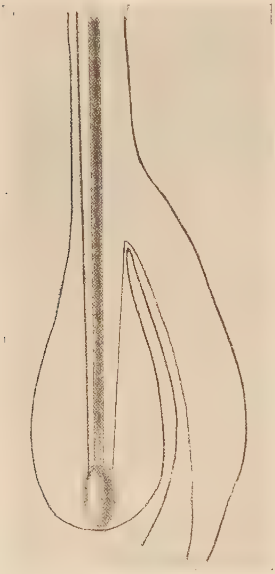

Fig. 15.—Sound pocketed in a diverticulum. Esophageal probe bulb threaded on a swallowed thread passed into diverticulum (Plummer).

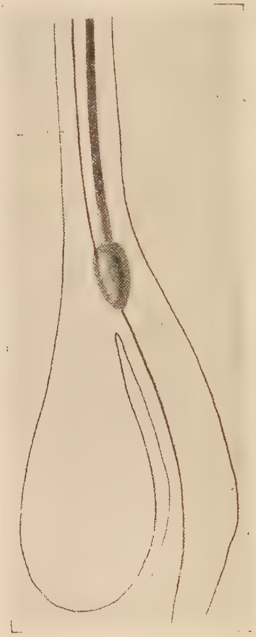

Fig. 16.—Traction on the thread lifting the sound out of the diverticulum. Probe bulb elevated by tightening the thread, showing depth of sac (Plummer).

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[1] Trans. Sect. Surgery, Amer. Med. Assoc., Sixty-first Session, St. Louis, June, 1910; also Jour. Amer. Med. Assoc., February 25, 1911.

also its depth, by the amount of elevation of the probe on tightening the thread. By means of the thread as a guide it is possible to pass through apparently nearly impermeable strictures with a small olive and gradually to dilate them. Only sufficient force to detect an obstruction and readily to pass through it with olive bougies of smaller size is permissible with an unguided sound. With the silk thread as a guide, sufficient force may be used short of carrying the thread out of its course and through the esophageal wall. If sufficient obstruction is present to make it possible that the thread might be carried out of its course, a piano wire (Fig. 17, *e*) should then be passed on the thread, and then the olive passed on the wire. This renders, in some cases, the sound more easily introduced. When passing the olive over the wire, tension on the thread should be maintained to avoid any possibility of the tip of the wire injuring the esophagus or stomach wall. In Fig. 18 and Fig. 17, *d* are shown the distal portion of the piano wire guide and carrier for working the wire through a tortuous cicatricial stenosis on a thread. Various sizes of piano wire may be used and its flexibility may be varied by the amount of wire exposed.

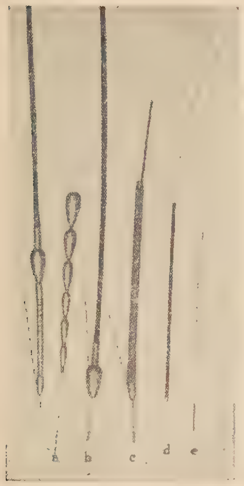

Fig. 17.—Various forms of esophageal sounds (Plummer).

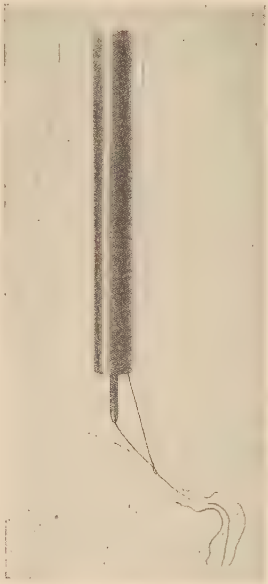

Fig. 18.—Piano-wire guide and carrier (Plummer).

In the treatment of cicatricial stenosis, the wire guide is not often necessary after the first dilatation, and the thread may be dispensed with as soon as the smaller sizes of spindles will readily pass. Plummer recommends further dilatation with a sound (Fig. 17, *a*, *b*), consisting of a series of superimposed spindles (olives), the smallest one being at the bottom and gradually increasing in size. Lerche[1] has devised a flexible wire shaft esophageal sound, covered with rubber, on the ends of which olives of various sizes can be screwed. They are perforated so that the sounds can be threaded on a guide.

A stomach-tube with a perforated metal tip can be employed over a thread as a director, the tube being stiffened with a whalebone staff in order to obtain the contents of a diverticulum, or, as in Fig. 19, a case of diffuse dilatation of the esophagus, to enter the stomach. By this method it is possible to feed the patient while gradually dilating the stenosis, and thus avoid in some cases a preliminary gastrostomy.

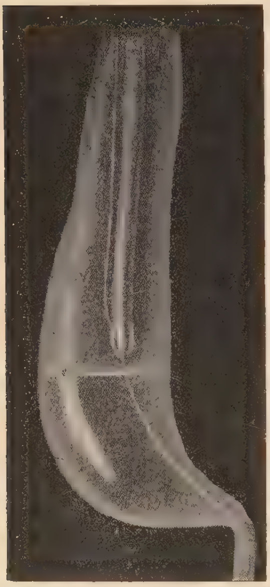

Fig. 19.—Metal-tipped stomach-tube guided by a thread (Plummer).

In endeavoring to calibrate a stricture in the lower 3 inches of the esophagus, or to dilate the same by means of a sound, one must remember the following anatomic features: the thoracic portion of the esophagus lies nearly in the median line, but deviates to the left as it passes forward to the opening in the diaphragm. In passing an olive through the lower 3 inches of a normal esophagus the staff impinges on the left anterior wall and the olive is directed to the cardia by the left posterior wall. This last portion presents an increasing degree of obstruction as it approaches a *horizontal plane* in cases of diffuse dilatation. The degree of obstruction encountered depends also on the stiffness of the staff, the size of the olive, and the tone of the wall of the esophagus. A stiff staff (Fig. 20, A) would have a tendency to injure or penetrate the wall, while a flexible staff, B, would curve in conformation to the shape of the esophagus and would approach the opening at an angle with this portion of the esophagus and no harm result.

In some cases, however, of *diffuse dilatation of the esophagus* from cardiospasm an unguided sound cannot be passed with safety, but a 15 mm. olive will readily and safely pass, when guided by the thread.

The flexible sound or a small stomach-tube is more readily passed and with greater safety in the case of a carcinomatous cardia (Fig. 21).

*Benign Stricture.*—A benign stricture may be dilated every day, or every other day, or according to indication. The *use of olive dilators* or soft tubes, when possible, *is preferable.* Soft-rubber bags of various design, introduced empty and then inflated, have been advocated. Metal dilators which can be expanded by a screw thread and wheel, modeled somewhat after urethral dilators, have been devised, and Lerche[1] recommends an esophageal dilator, which is used through the esophagoscope under guidance of the eye, somewhat like the Kollman dilator.

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[1] Jour. Amer. Med. Assoc., July 29, 1911.

*Malignant Stricture.*—The history and age of the patient, absence of the Wassermann reaction, and cachexia, will usually determine malignancy. A palpable tumor may sometimes be felt if the stenosis is above the sternum and also enlarged glands are present. In such event the author deprecates the attempted passage of an instrument through a cancerous stenosis.

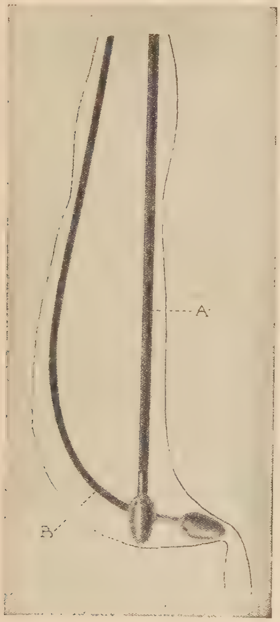

Fig. 20.—Diagram of diffuse esophageal dilatation, showing danger from stiff staff (A), together with use of flexible staff (B) (Plummer).

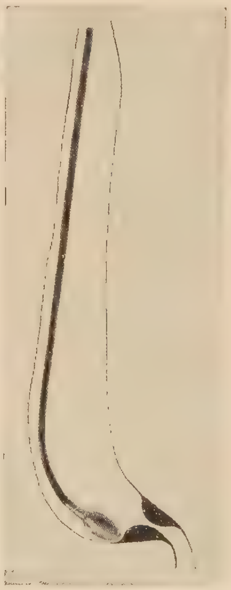

Fig. 21.—Passing a sound through a carcinomatous cardia (Plummer).

The esophagoscope is of chief value in locating foreign bodies and affording direct means of removal.

In the use of the sound one must remember that it is 6 inches from the incisors to the commencement of the esophagus at the cricoid cartilage; 9 inches to the crossing of the left bronchus, and 16 inches to the cardiac orifice of the stomach. There is normally some constriction of the esophagus at these three points.

Obstruction to the passage of the tube may be due to esophageal spasm, but by waiting this will usually subside. I have found that in some of these cases a large tube can be passed more readily than a small one. If the tube passes readily on one occasion and refuses to pass on another, it has probably slipped into a diverticulum, or the latter has filled up and by pressure prevented the passage of the instrument.

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[1] Jour. Amer. Med. Assoc., July 29, 1911.

If the obstruction is permanent, one must decide whether it is due to stricture (narrowing) or external pressure, such as from aneurysm, tumor, or enlarged glands. Syphilis, cancer, tubercular, peptic, and rarely typhoid ulcers, and contraction following burns from acids or alkalis are the principal causes of stricture. Congenital stricture is rare and is usually found in the upper part of the gullet.

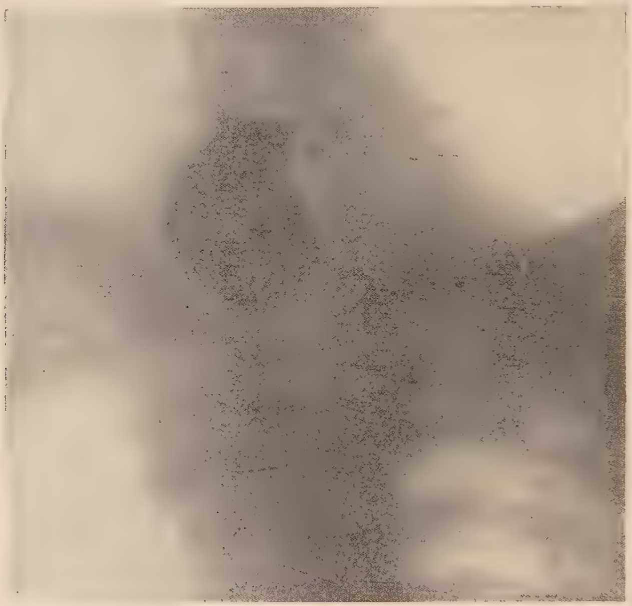

Fig. 22.—Diverticulum of esophagus, from skiagraph (Plummer).

If stricture be present, the locality, caliber, and permeability must be determined.

*Locality.*—Pass the tube or olive to the strictured point, nip it close to the incisor teeth, and measure the distance from here to the entering tip of the tube or olive after withdrawal.

*Caliber.*—Sounds or olives of varying diameters will determine the caliber by finding one which will pass the obstruction. By the esophageal bruit and the use of a somewhat rigid sound one can tell whether or not the obstruction is *permeable.*

Occasionally small portions of new growth may be found in the openings of a fenestrated tube. Blood shows ulceration or erosion.

*Contraindications.*—The tube should never be passed if there is aneurism of the thoracic aorta or recent vomiting of blood.

**X-ray Examination.**—From a practical standpoint, stenosis of of the esophagus can be readily diagnosed by the various methods of sounding, and a diverticulum by the symptoms already described, and by Plummer's thread method as a guide, to which the writer has already referred. Röntgenography will disclose thoracic masses obstructing the esophagus by outward pressure and occasionally an esophageal tumor. An impacted foreign body can also be thus determined. The position, shape, size, and relations of diverticula and dilatation are also shown when filled with bismuth mixture. Diverticula are thus demonstrated in Figs. 22 and 23.

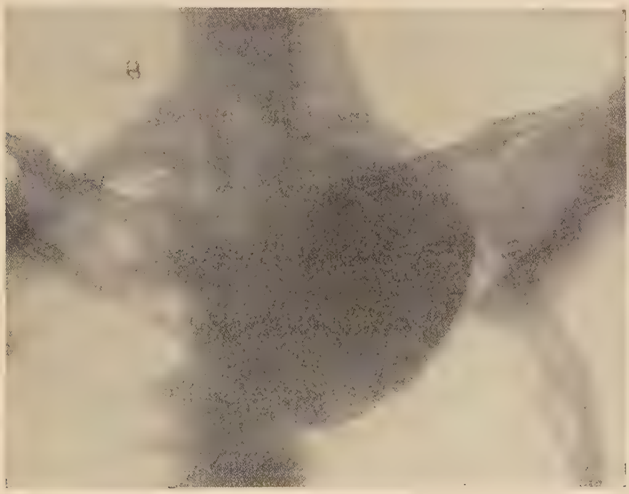

Fig. 23.—Diverticulum of esophagus, from skiagraph (Plummer).

Large dilatations occurring in the upper third of the esophagus above organic strictures usually have a *tail-like portion* of bismuth extending down from the bottom of the sac, thus distinguishing them from diverticula. When the tail is absent, and there is doubt, Plummer's thread guide method will differentiate the two conditions.

With *diffuse dilatation of the esophagus*, following spasmodic and infrequently organic stricture, at the cardia, the shadow is large and irregularly spindle shaped.

With cardiospasm, the lower extremity of the shadow is cone-shaped and its apex corresponds to the hiatus esophagi (Fig. 24). With organic stricture, with diffuse dilatation, the outline is irregular and terminates above the hiatus. In most cases the ditatation extends to the upper border of the manubrium and is constricted at the root of the lung. Dilatation occurring above organic strictures are, as a rule, of limited extent and irregular in outline in their lower portion.

Mucilage of acacia is recommended as a valuable vehicle for the bismuth mixture.

Other methods of differentiating a dilatation of the esophagus and a diverticulum have been described,[1] but those to which the author has referred are the most practical.

The writer briefly suggests the following methods of treatment:

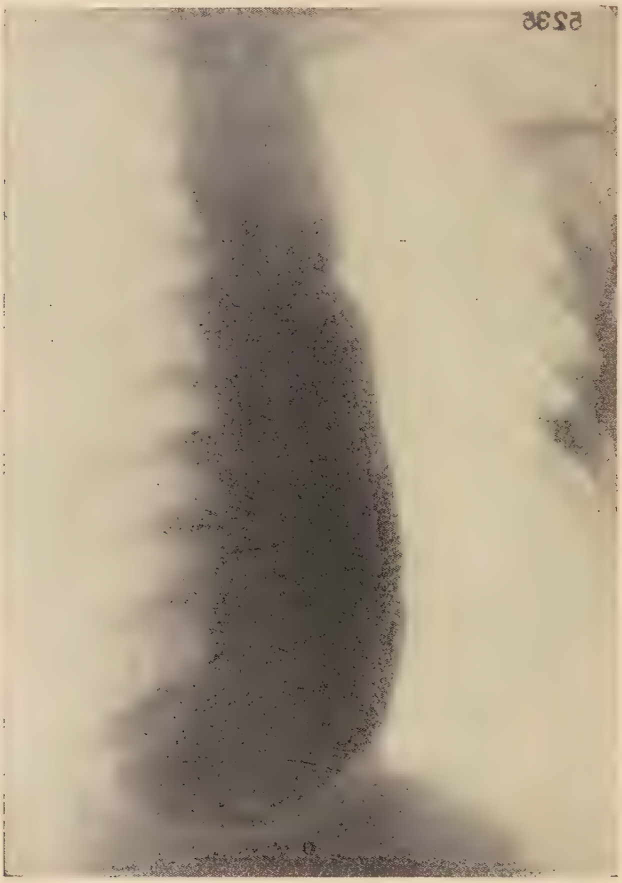

Fig. 24.—Diffuse dilatation of the esophagus in case of cardiospasm (Plummer).

For benign stricture or cardiospasm with dilatation of the esophagus, soft-rubber inflating bags and mechanical dilators, opening their jaws by manipulating a screw thread from above, have been advocated. The use of various sized olive dilators, combined, when necessary, with the thread guide, as devised by Plummer, seems most practical.

At times internal section of the stricture can be made by manipulation through an esophagoscope, or, on occasions, a preliminary gastrostostomy, with the subsequent thread-cutting method of Abbé, may be employed. Willy Meyer[1] reports a successful case of impermeable cardiospasm treated by thoracotomy and esophagoplication.

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[1] Kelen, New York Med. Jour., Feb. 23, 1907.

Syphilitic cases require appropriate treatment—" 606," etc.

Thiosinamin (0.06 to 0.12) has been recommended hypodermically every two to three days as an aid to the treatment of benign strictures.

Liquid diet is indicated and lavage of the diverticulum or dilated esophagus during medical treatment. In the case of malignant stricture, radical operation may occasionally be possible if the stenosis is high up and there is no great involvement.

Preliminary gastrostomy is indicated in such an event. Gastrostomy is indicated for the purpose of prolonging life in all malignant cases. For the cure of diverticula operative procedure is usually required.

**Acute Esophagitis.**—Mechanical, chemical, or thermal irritation (too hot liquids) are the chief causes. It may also accompany acute infectious diseases or result from extension from adjacent tissues.

*Types.*—It may be catarrhal, follicular, ulcerative, phlegmonous, or necrotic.

*Symptoms.*—With corrosive poisons inspection of the throat and pharynx often makes the case clear, and there may be vomiting of necrotic mucosa streaked with blood. Pain, dysphagia, and thirst are present in most cases, with expectoration of mucus in the milder types. Pain is increased on swallowing dry or hard food or very hot or cold fluids. Instrumentation is painful; occasionally perforation may take place in the phlegmonous or necrotic types.

*Treatment.*—Antidotes and emollients are indicated in the cases due to poisoning, and also immediate lavage. Olive oil with bismuth in suspension is of value, and hot or cold external applications. Small bits of ice dissolved in the mouth may furnish temporary relief. Rectal feeding may be temporarily indicated. Rarely a hypodermic of morphin or codein may be required. The occasional use of a 2 per cent. cocain or eucain spray may render swallowing less painful.

**Chronic Esophagitis.**—This type of esophagitis occurs most frequently in alcoholics or secondary to cardiac or pulmonary disease of long standing. It may follow an acute esophagitis or be secondary to an inflammatory condition occurring with carcinoma of the esophagus, dilatation, or a diverticulum. Thrush may also extend into the esophagus.

*Pathology.*—There are grayish streaks of degenerated epithelium on the summit of the longitudinal folds and an increase of mucus. The mucosa may be dusky red or bluish and have a granular appearance, with local areas of desquamation or ulceration.

*Symptoms.*—There may be uneasiness or difficulty in swallowing and a feeling of distress behind the sternum, which persists after irritating food or drink. The symptoms may be masked by those of the primary disease.

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[1] Jour. Amer. Med. Assoc., May 20, 1911.

*Treatment.*—The main indication is removal or treatment of the cause. Alcohol, spiced foods, acids, and hot fluids are to be avoided. Demulcents and soft food are indicated. Occasionally, local application of a weak solution (1 per cent. of silver nitrate or tannin) may be of value. Bismuth subnitrate, 30 grains (0.2), suspended in 1 to 2 ounces of olive oil, three times a day, is useful when there are erosions.

**Ulcers of the Esophagus.**—*Etiology.*—Severe burns from the ingestion of acids or alkalis, syphilis, typhoid, cancer, and tuberculosis may produce local ulceration of the esophagus. Decubital ulcers may occur in emaciated persons who have suffered from wasting disease. They usually are found at a level with the cricoid cartilage, resulting from pressure, probably of the esophagus, between the larynx and vertebræ when in the dorsal position. Pressure ulcers may result from circulatory interference of growths encroaching on the esophagus. Uremic ulcers occasionally occur and peptic ulcers are probably the most common.

The latter possibly result in part from regurgitation of the gastric juice. The peptic ulcer of the esophagus resembles that of the stomach. In some cases it lies just above the orifice of the cardia, while in others it begins in the stomach and extends through the cardia into the esophagus.

*Symptoms.*—In some cases esophageal ulcer is latent during life, while in others there are no symptoms until perforation takes place. Dysphagia and hemorrhage may occur. If the ulcer lie well up in the esophagus, there may be local pain on swallowing at the site of the ulceration; while with the peptic type the pain lies behind the sternum or at the level of the ensiform. It may radiate to the back. The pain usually occurs on swallowing or immediately afterward, and is thus earlier than with gastric ulcer.

*Diagnosis.*—During active hemorrhage no instrumentation should be employed. About ten days later the gentle passage of soft stomach-tubes of various size may enable one to determine the sensitive area of the ulcer. Olives of various sizes with a flexible stem may be cautiously employed for the same purpose. In some cases one may examine carefully with the esophagoscope without the mandrin, with the light turned on, as in the case of removal of foreign bodies. Some recommend locating the ulcerated area by the thread method. The patient swallows about two feet of No. 15 braided surgeon's silk, the end being tied about the patient's ear. This is left in situ for two to three hours and then withdrawn. Blood will stain the thread where it passes over the ulcerated surface, and its distance from the mouth can thus be located.

This method for esophageal ulcer is much more accurate than when employed for gastric ulcer, for obvious reasons. It is worthy of trial in the former, but the writer does not believe it of value for the diagnosis of duodenal or gastric ulcer.

**Perforation of the Esophagus.**—Intense thoracic pain, dyspnea, and collapse are the salient symptoms. Pneumothorax or hydropneumothorax may follow. If perforation occur high up in the neck, local inflammation may result. Perforation may also take place into the mediastinum. If perforation occurs below the diaphragm, subphrenic abscess or peritonitis may follow.

*Treatment.*—For hemorrhage, morphin, $\frac{1}{8}$ to $\frac{1}{4}$ grain (0.008–0.016), should be given by hypodermic injection. Adrenalin chlorid (1 : 1000), 5 to 10 drops in $\frac{1}{2}$ to 1 ounce of water, is to be administered by mouth for the local styptic effect. Gelatin solution or Trémolière's solution, as described under Gastric Ulcer, should also be given. A piece of cracked ice may be swallowed and an ice-bag applied externally.

Rectal feeding should be employed for a few days, and subsequently liquid diet for several weeks. The patient should remain in bed. Bismuth subnitrate in 30-grain (0.2) doses should be begun on the second or third day in 1 ounce of water by mouth, four times daily.

Tincture of belladonna, 10 drops, four times a day, in 1 dram (4.0) of water can be given for the spasmodic pain. If the ulceration be syphilitic, as shown by the Wassermann reaction, "606" or appropriate antiluetic treatment should be instituted. Tubercular ulcer may receive local treatment through the esophagoscope and at times ulcers of other types. A bougie should occasionally be cautiously passed to prevent stenosis. If symptoms of the last occur, treatment should in any event be at once begun.

If recurrent hemorrhages occur or stenosis becomes progressive, a gastrostomy is indicated. The stricture should be ultimately divided. If perforation occurs, operation is indicated. Its character depends on the location of the perforation.

Gastrostomy is also indicated in connection with the emergency operation, in order to give rest to the esophagus and thus enable one to feed the patient.

**Stricture of the Esophagus.**—The method of determination of stricture of the esophagus, malignant, benign, and that due to cardiospasm of persistent type, with resulting fusiform dilatation of the esophagus, has previously been described.

**Carcinoma of the Esophagus.**—Cancer may occur in any part of the esophagus, with resulting stenosis and infiltration of the neighboring structures.

In the English-speaking countries, statistics show that cancer of the esophagus is only slightly less frequent than carcinoma of the stomach. The disease is most frequent in the male and usually occurs between the ages of forty and sixty years.

The squamous-celled epithelioma is the most common, or the adenocarcinoma. Occasionally the growth is of the soft, fungating, cauliflower type. Stenosis results with dilatation of the esophagus above the point of stricture.

*Symptoms.*—In a patient over forty years of age, in whom there is no history or any cause that might produce a cicatricial stricture of benign type, where dysphagia (difficulty in swallowing food) gradually develops, when stenosis is determined by examination and the patient is rapidly losing weight and strength, carcinoma is properly the diagnosis. Regurgitation of food is present, and the higher the obstruction, the sooner it occurs. The food regurgitated or aspirated is alkaline and contains no gastric secretion. Blood (visible or occult) and also pus are present later when ulceration has occurred. One of the recurrent laryngeal nerves may become involved and there may be dyspnea and a hoarse voice. If the sympathetic system is affected, there may be contraction of the pupil on that side, with ptosis and sluggish reaction to light. There is a feeling of pressure and sometimes severe pains are present near the seat of the growth or radiating into the shoulders and back. Cachexia and marked anemia develop. Occult blood may be found in the feces. The cervical and supraclavicular glands are often palpable later in the course of the disease. The growth with enlarged glands, if it be in the neck above the level of the sternum, is palpable quite early.

The growth usually extends into the surrounding tissues, involving eventually the blood-vessels, nerves, pleura, lungs, diaphragm, pericardium, mediastinum, and glands.

The method of examination for all types of stenosis has already been described. Syphilis should be excluded by the Wassermann and Noguchi tests.

*Treatment.*—Gastrostomy is indicated and later complete excision of the growth, if such be possible. Gastrostomy in any event allows feeding the patient through the fistula and thus prolongs life.

If *consent cannot be obtained for operation*, lavage of the esophagus above the stricture, liquid diet, cocain (2 per cent. spray), or eucain (3 per cent. spray) before eating, and hypodermics of morphin, $\frac{1}{8}$ to $\frac{1}{4}$ grain (0.008–0.016), to relieve pain, are indicated. The ingestion of olive oil, $\frac{1}{2}$ to 1 ounce before each feeding, aids the passage of food through the stricture, just as it is of value in pyloric stenosis for the same purpose. Local treatment with radium and cautious dilatation may be tried.

**Benign Growths of the Esophagus.**—These are rare and usually of small size, such as fibroma, papilloma, myoma, fibromyoma, and lipoma. They may be single or multiple, pedunculated or sessile. The symptoms are those of a mild stenosis. The esophagoscope will determine their presence and in some cases they can be removed through it by a snare.

**Examination of the Abdomen.**—The special methods of physical examination of the stomach and intestines are described in the parts of this volume devoted to these subjects. It seems advisable to refer to the general methods of examination of the abdomen and the other viscera.

*Anatomic Landmarks.*—The ensiform appendix and down-curved arches of the ribs constitute the upper bony landmarks. The iliac crests, anterior superior spines of the ilia, and the symphysis pubis in the median line are the lateral and lower boundaries.

The linea alba lies between the recti muscles in the median line, runs from the ensiform appendix to the pubic symphysis, and is visible as a groove above the umbilicus.

The umbilicus is somewhat variable in its position, though usually lying about 2 inches above the bispinal line drawn transversely between the anterior superior spines of the ilia. The recti muscles lie on each side of the linea alba and are bounded externally by the lineæ semilunares, which run with an outward curve from the lowest part of the seventh rib to the pubic spines. These lines lie on each side about 3 inches from the umbilicus.

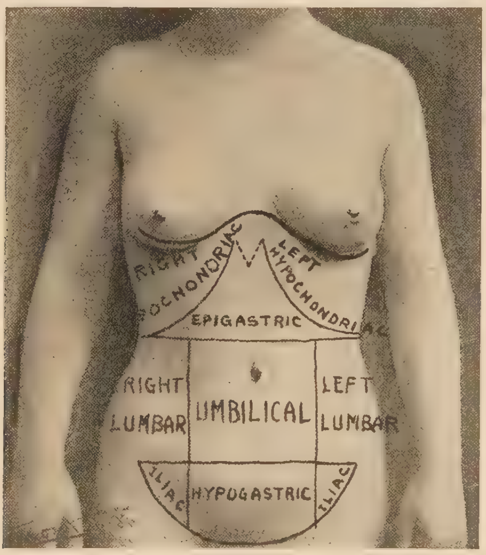

Fig. 25.—Topographic areas of the abdomen.

*Topographic Areas.*—In order to describe the situation of organs or lesions, the surface of the abdomen is divided into regions, of which the method depicted in Fig. 25 is in general use.

There are nine regions divided off by four lines, two horizontal and two vertical. The horizontal lines are the infracostal or subcostal, drawn transversely at the level of the inferior borders of the tenth ribs, and the bispinal line, connecting the anterior superior spines of the ilia. The vertical lines pass through the center of Poupart's ligament on each side and are downward prolongations of the mammillary lines of the thorax. The boundary lines between the epigastric and hypochondriac regions correspond with the costal margins, and the iliac regions correspond to the so-called inguinal regions.

A second method of dividing the abdomen is by a vertical and transverse line through the umbilicus, forming four quadrants.

In reference to the various landmarks, one often measures distances by the finger-breadth. The average finger-breadth is $\frac{3}{4}$ inch; two, $1\frac{1}{4}$ inches; etc.

A hand-breadth averages $3\frac{1}{2}$ to 4 inches.

*Preparation of the Patient and Technic.*—The patient should preferably lie in bed in the dorsal position, quite flat, with the head on a single thin pillow. The same posture should be assumed if the examination be made on the office table. The bedclothing should be drawn well down, except the sheet, under cover of shich the nightdress or undershirt should be drawn up to the lower sternum, and the sheet then folded down to a short distance above the pubes. The sheet may not be required in the office, though generally preferable.

The patient should lie as symmetrically as possible, with the pubic spines at the same level and a good light secured. It is of value to make an observation also in the *sitting* or *standing position*, as *prolapse of the abdominal walls* or *viscera* can thus be more clearly seen.

*Inspection.*—Inspection should be from the front, sides, and back. It is of great importance. With excessive abdominal distention the skin is smooth, shining, and stretched. Copper-colored, scaly, somewhat circular spots are significant of secondary syphilis; and whitish streaks, or striæ, of long-continued distention, such as from pregnancy or ascites.

Typhoid eruption or exanthemata may in some cases be in evidence. Glandular enlargements in the groin or old scars are suggestive of venereal infection. Inguinal, umbilical, or femoral hernia may be observed.

Inspection of the blood-vessels often affords valuable information. Enlarged veins radiating from the umbilicus (the caput medusæ) are significant of portal obstruction, cirrhosis, or tumor of the liver. General enlargement of the abdominal veins may be present in similar conditions, or from pressure on the venæ cavæ by thoracic or abdominal tumors. If a dilated lateral vein is present running up the right midaxillary line, it should be emptied by massage, and the *method of its refilling* carefully observed. If the portal vein, or the inferior vena cava, is obstructed, the direction is upward; but if the superior vena cava is pressed upon, the direction is downward.

Distention of the veins in the pubic region alone shows some probable obstruction below the liver.

Enlarged epigastric arteries are diagnostic of obstruction of the aorta or iliacs.

An umbilicus that protrudes is suggestive of hernia, ascites, pregnancy, or some form of abdominal distention.

Absence of respiratory abdominal movements, with accentuation of thoracic respiration, is significant of peritonitis.

Peristaltic unrest (visible peristalsis) is usually diagnostic of stenosis of the pylorus or intestines or of intestinal obstruction. It may rarely occur normally in thin persons.

One can at times infer the site of the obstruction by the location and character of the peristalsis. The waves run in the stomach from left to right and in the transverse colon from right to left. If the obstruction is near the ileocecal valve, the *swollen* and *moving coils* of the intestine lie one above the other in the central part of the abdomen (ladder pattern).

If the constriction is lower down in the large intestine, the distention is chiefly visible in the course of the colon (in the circumference of the abdomen). A recurring protuberance at one point, disappearing with a loud sound, is probably near the point of stenosis.

*Protrusion from a tumor* can at times be observed.

*Method of Abdominal Palpation.*—The right hand should be warmed and laid flat upon the surface of the abdomen, the physician sitting to the right of the patient (Fig. 26).

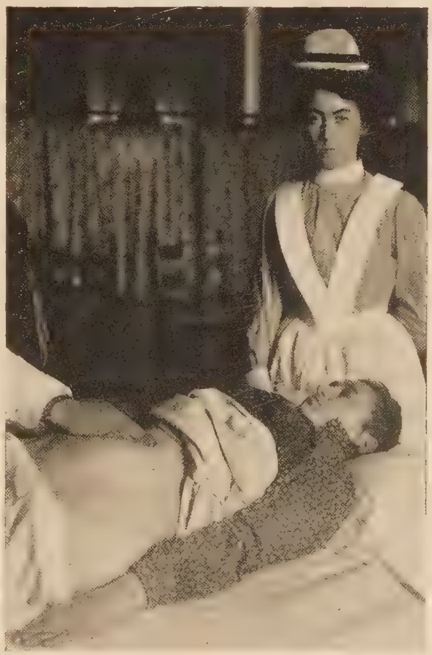

Fig. 26.—Abdominal palpation.

Palpation should be at first with somewhat circular pressing movements, sliding the skin over the parts beneath, and passing from one portion of the abdomen to another. One should not poke suddenly with the finger-tips. Gradually deeper localized palpation may be made with the finger-pulps to determine the presence of tender spots, or the shape, size, and mobility of existing masses or swellings.

The facial appearance of the patient rather than the verbal expression tells whether true pain is present. Tenderness suggests inflammation or ulceration. McBurney's point should always be explored for appendicitis.

If firm pressure elicits tenderness, it is apt to be real and deep seated, rather than a surface lesion or hyperesthesia.

If hysteria is suspected, the patient's attention can be diverted by pressing on a different part of the surface with one hand, while the other hand explores the original seat of pain complained of. Absence of true tenderness is thus frequently revealed.

If the abdominal muscles are contracted, the knees and thighs should be flexed and a pillow placed beneath the head and shoulders to secure relaxation. The *flexion of the lower limbs* I believe *preferable in every case.* Deep and rapid respirations at the end of expiration relax the muscles momentarily and render examination more easy. This method aids in differentiation, whether the mass felt is due to contraction of the belly of a muscle, such as of the rectus, and whether a tumor is movable with respiration. Howard Kelly recommends vibratory movements with the fingers while palpating.

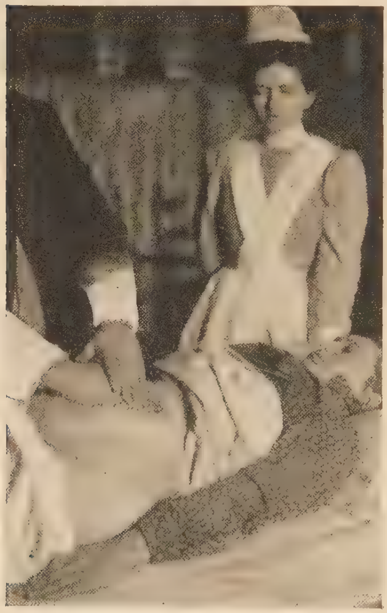

Fig. 27.—Reinforced palpation.

In some cases reinforced palpation, the left hand exerting pressure over the right, as in Fig. 27, is of value, especially in examination of the deeper organs. During examination forced respiration should be taken, and at each expiration the abdominal wall should be pressed upon firmly, maintaining during inspiration the ground which has been gained. This method is of special service in determining a chronic enlarged appendix

If there is fluid in the peritoneal cavity and one desires to palpate an organ which is obscured by its presence, sudden deep pressure with the finger-tips ("dipping") will displace the fluid. In some cases a general anesthetic may be necessary for a thorough examination.

All the regions of the abdomen should be explored, the *umbilical,* the *inguinal,* and *femoral regions* being examined for hernia.

When the lateral regions are examined, both hands should be employed, one being slipped under the body so as to make forward pressure between the last rib and iliac crest, thus pushing forward the structure against the examining hand in front.

At times it may be of service to examine in the knee-chest position, or with the patient standing and leaning forward, supporting himself with the hands by a table or chair. If he is very fat, it is often useful to have him turn partly on the side, thus "spilling" the intestines and fatty abdominal walls away from the region under investigation.

*Digital rectal* and *vaginal examination* are very necessary in many cases, especially when the lesion is situated in the lower third of the abdomen.

There is a *method of abdominal examination* used abroad which I have not seen recommended in our text-books. This is the practice of *making abdominal*, or *bimanual*, vaginal examination of the *patient while in a very hot bath.* In many instances the abdominal relaxation thus obtained nearly equals that while under an anesthetic, with the additional advantage that the patient can help the examiner by voluntary movements, such as deep inspiration, holding the breath, etc. The examination can be made in an ordinary bath-tub in water as hot as the patient can bear. If it is necessary to have the patient higher in the tub, a long sheet can be let down over the tub into the water and fastened about the ends of the tub by knotting the corners under the rolling edge, or by passing clothesline beneath the tub. The patient is thus suspended in a hammock.

Rigidity of one or both of the recti muscles is of great diagnostic importance, being significant of peritoneal irritation, local peritonitis (if one muscle be involved), or general peritonitis if both recti and all the abdominal muscles are affected.

Occasionally a rigid rectus is found on the side of a pneumonia or diaphragmatic pleurisy.

The upper segments of one or both recti may be rigid in abscess of the liver, or in subphrenic abscess, or of the right rectus in acute cholecystitis; the right rectus, especially the lower segment, in appendicitis; and the left rectus in diverticulitis, or in left-sided pelvic inflammation.

Mensuration of the circumference of the abdomen at the level of the umbilicus and of the length of its anterior wall from the ensiform to the symphysis, are of use in noting the increase of ascites or the growth of a large tumor. An uneven protuberant surface is characteristic of a malignant growth; an even surface is more often found with benignant neoplasms or intussusception. A fecal tumor can usually be indented, and as the finger is raised the intestinal wall slips from the mass.

*Percussion of the Abdomen.*—With the exception of pulmonary resonance, which we note in defining the upper limits of the stomach and liver, and splenic and hepatic dulness, the normal abdomen is tympanitic. From the presence of food in the stomach or fecal accumulation in the intestines there are variations, with resulting dulness or even flatness. The percussion note over the sigmoid flexure and lower part of the descending colon is quite frequently dull, owing to the tendency to fecal accumulation in these regions.

In general, we may say that the pitch of the resonant note varies with the size of the air space and the degree of tension of the containing cavity; the smaller the air space and the greater the tension, the higher is the pitch. Hence, the empty stomach and colon would afford a lower pitched note than the small intestine.

The presence of food and liquid in the stomach modifies the results of percussion, as do also feces in the large intestine. For example, with an empty stomach we have tympanites; and then quite frequently a change in note over the transverse colon to dulness or even flatness; or with the partially full stomach and empty intestine, tympanites above, then dulness or flatness over the contents and intestinal tympanites below. It is well, therefore, to have the large intestine cleared out by enema before examination. Practically we find in many cases stomach tympanites with change in note over the colon due to some contents.

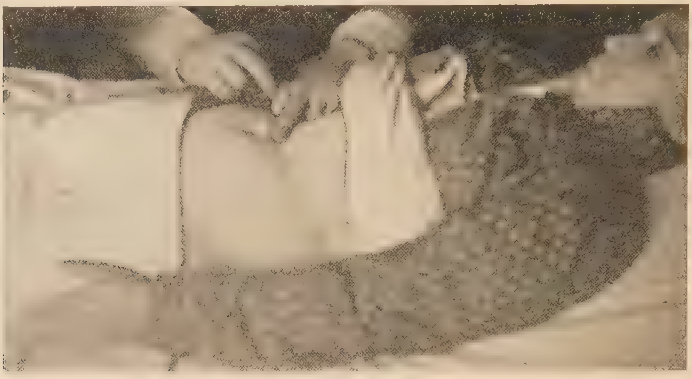

Fig. 28.—Simple percussion.

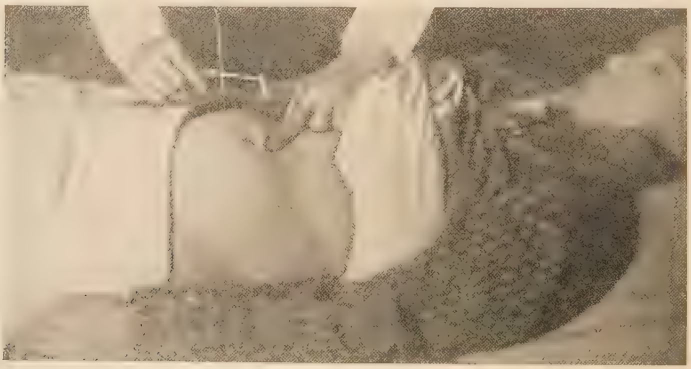

Fig. 29.—Percussion hammer.

Among the best methods of percussion are simple percussion with the finger or hammer, flicking percussion, auscultatory percussion, and the " scratch method " of auscultation.

In simple percussion, the middle finger of the left hand should be laid flat on the abdomen (the pleximeter) and the middle finger of the right hand, bent at right angles, should be employed as the plexor, as depicted in Fig. 28. The other fingers and thumb should be folded into the palm of the hand.

In Fig. 29 is shown the method with the percussion hammer and the correct position. The finger is preferable as a pleximeter, as the rubber instruments interfere with the sounds.

"Flicking percussion" is useful in detecting slight degrees of dulness.

The forefinger or middle finger of the left hand should be placed nail downward on the surface; the middle finger of the right hand is well flexed, so that the nail is pressed against the palmar surface of the thumb. It is then suddenly allowed to escape, so as to strike sharply against the palmar surface of the finger lying on the abdomen (Fig. 30).

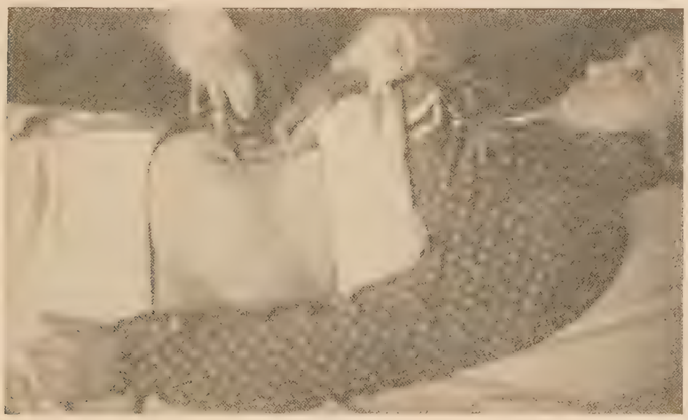

Fig. 30.—Flicking method of percussion.

Auscultatory percussion is probably of greatest value in *outlining contiguous air-containing viscera.*

The stethoscope is placed over the organ and the normal note secured by percussion close to the instrument. Then percussion is carried out, beginning at some distance, from above, below, and laterally, and the change of note observed.

This method and the "scratch" method will be described later in outlining the position of the stomach and intestines.

If the percussion note of a deep-seated mass is to be elicited, the pleximeter finger must be pressed slowly and firmly down in order to push aside or compress air-coils of intestine which would mask the note.

If dulness is present where it should not exist, it should be ascertained whether it disappears or shifts with changes in the position of the patient, *i. e.*, whether it is fluid.

If the distention is due to ascites (fluid), the center of the abdomen is flattened and the lateral and dependent portions bulge outward, providing the fluid is not excessive. If it is very great, the abdomen is arched and prominent, the umbilicus is bulging or stretched, and the shape is not changed when the posture is altered.

On percussion, the flanks are dull and the center of the abdomen tympanitic, as the intestines float to the highest point. Unless the quantity of fluid is excessive the line of dulness changes its position, as the patient is turned on the side, the fluid gravitating to the lowest point and being replaced by the tympanitic intestine. The upper flank, previously dull, is now tympanitic. If a small amount of fluid is suspected, percussion in the umbilical region in the knee-chest position will give dulness when it was tympanitic in the dorsal position.

Fluctuation may be elicited if there is considerable fluid.

The ulnar edge of a nurse's or assistant's hand should be pressed firmly on the linea alba, to cut off muscular vibrations.

One hand of the examiner is placed upon one lateral abdominal wall, while he should tap sharply with the fingers on the opposite side. If fluid is present, a transmitted wave—at times visible—will be felt by the palpating hand.

With tumors, enlargement of the abdomen is not symmetric. Percussion does not show the uniform resonance of gas nor the lateral dulness and central tympanites of fluid, and palpation demonstrates the solidity of the mass.

With gas, the abdomen is arched and tense, universally tympanitic, and fluctuation cannot be obtained.

*Sources of Error.*—The segments of the recti muscles when contracted may simulate a small tumor.

By insinuating the tips of the fingers under the edge of the apparent tumor and having the patient raise the head and shoulders, the muscle is felt to contract and thicken.

A localized contraction of the abdominal muscles or a persistent gaseous distention of a portion of the intestines (" phantom " tumor) may be deceptive. These occur, as a rule, in hysteric women and are dull or tympanitic, depending on the above conditions. They disappear during rapid forced respiration or under anesthesia.

Fitz believes that some " phantom " tumors are congenital or acquired dilatation of the colon.

**Auscultation of the Abdomen.**—*Sounds in the Abdominal Cavity.*—In the healthy intestines there are always bubbling or gurgling sounds heard on auscultation. The entire absence of sound is significant of intestinal paresis.

With mechanic obstruction the sounds are usually increased in intensity and number.

With intestinal paresis, usually due to peritonitis, the heart and respiratory sounds may be audible over the entire abdomen. This

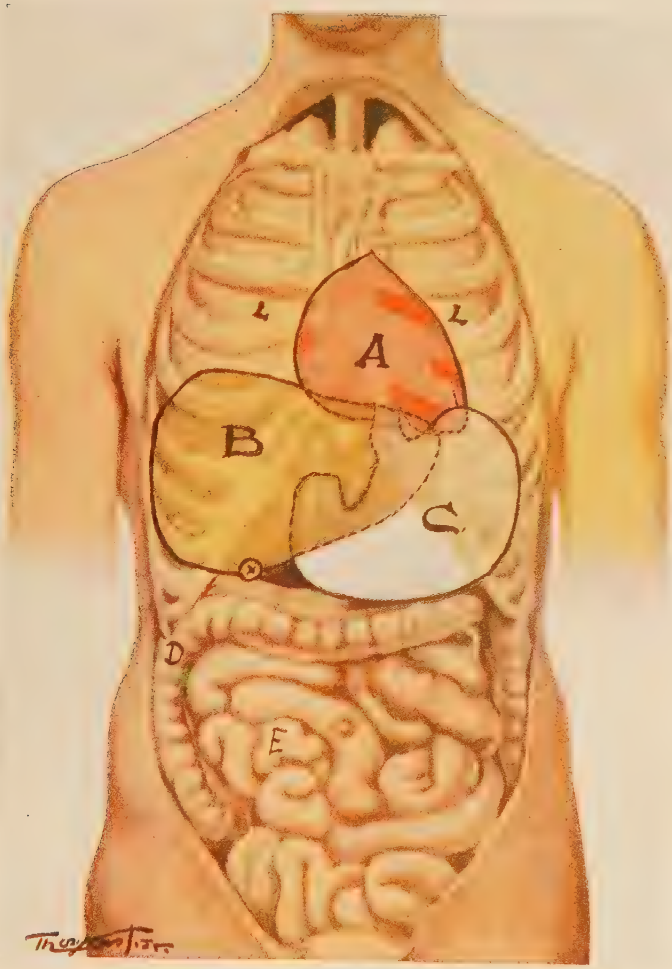

Fig. 31.—Relations of the viscera. Anterior view: *L*, *L*, Lungs; *A*, heart; *B*, liver; *C*, stomach; *x*, gall-bladder; *D*, colon; *E*, small intestine.

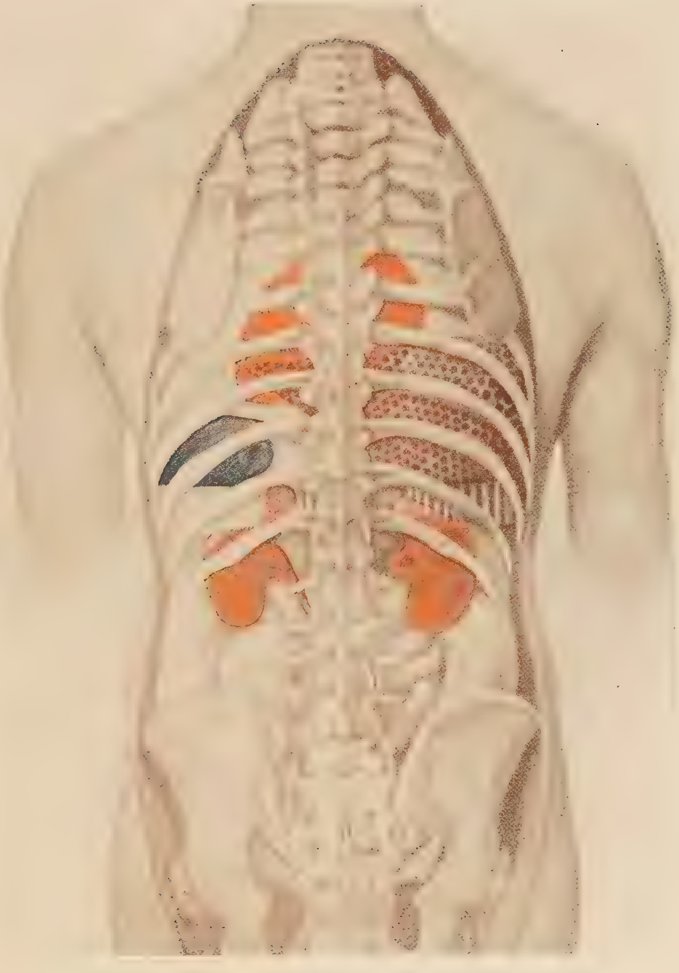

Fig. 32.—Relations of the viscera. Posterior view.

is not true in tympanites due to other causes. Crepitation or friction sounds are at times heard in peritonitis, as in perihepatitis in the right hypochondrium, or in the left hypochondrium with perisplenitis.

The venous hum or aneurysmal bruit of abdominal aortic aneurysm can be appreciated, or occasionally a venous hum over the liver from pressure on the vena cava. If pregnancy is present, there are the fetal heart sounds. The sounds over the stomach are of little diagnostic value, except the duration of the swallowing sound.

## TOPOGRAPHIC ANATOMY

The position and relations of the stomach and intestines have been described, but for the purpose of physical diagnosis we must briefly refer to the normal relations of the other abdominal viscera.

In Fig. 31 are depicted diagrammatically the relations of the organs on the anterior surface of the body, and in Fig. 32 the relations on the posterior surface.

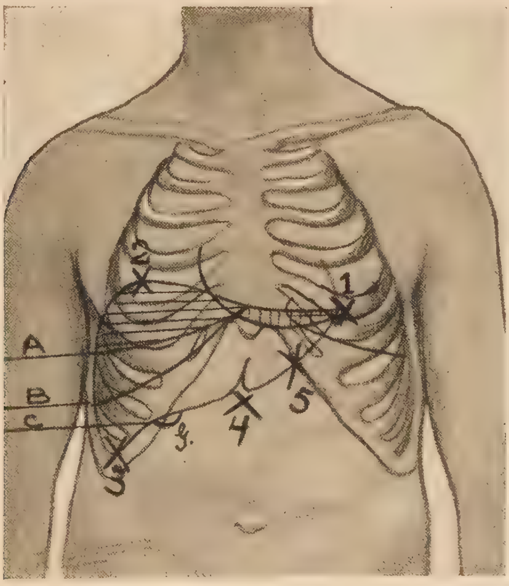

Fig. 33.—Delimitation of the normal liver (diagrammatic): *G*, Gall-bladder.

**The Liver.**—The general shape is that of a wedge with its base in the right hypochondrium, the upper surface lying in relation to the vault of the diaphragm, and its lower surface with the stomach, duodenum, gall-bladder, transverse colon, and small intestine; its anterior, lateral, and posterior portions are in relation with the abdominal parietes and lower right ribs.

*Delimitation of the Normal Liver.*—Mark a point 1 (Fig. 33) at the lower border of the fifth rib, between the left parasternal and mammillary lines, or about 2 to 2½ inches from the left edge of the sternum.

Point 2 lies in the fourth right intercostal space in the mammillary line. From 2 to 1 draw a connecting line, slightly convex upward on the right half and concave on the left, curving down at the lowest point to the base of the ensiform appendix.

From 2 draw a line nearly horizontally to the right and posteriorly, which should cut the midaxillary line in the seventh space and the scapular line in the ninth space, to the midspinal line. This line, front, side, and back, corresponds to the upper border of the liver.

To delimit the lower border, mark point 4 in the median line (lineä alba) about a hand-breadth (3½ to 4 inches) below the base of the ensiform process of the sternum.

Mark point 3 at the lower edge of the ninth right costal cartilage, and another point 5 at the edge of the left costal arch on a level with the lower border of the sixth rib. A line should then be drawn from 3 to 4 upward and to the left. At 4 is indicated the notch between the liver lobes. From 4 a slightly curved line to 1, passing through 5, should next be drawn. The line from 3 to 1 indicates the lower anterior border of the organ.

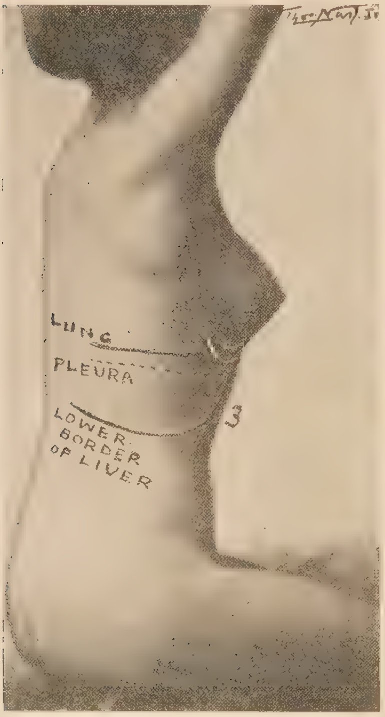

Fig. 34.—Lower border of liver (midaxillary line).

From point 3 draw a line backward and to the right, cutting the tenth intercostal space in the midaxillary line, from which point it joins the spine at a level of the eleventh rib (Fig. 34). This line demarks the lower border of the liver laterally and posteriorly, and is a continuation of the anterior inferior border.

This illustration shows diagrammatically the relations of the lower borders of the lung, pleura, and liver in the midaxillary line.

The left lobe lies to the left of the linea alba and extends nearly to the nipple, the notch lying in the midline. In the right mammillary line the liver extends from just below the level of the nipple to the costal margin.

The horizontal shadings in Fig. 33 show the portion of liver overlapped by lung, and the vertical shadings, that overlapped by the heart.

The gall-bladder, which is pear shaped, lies just internal to the ninth right costal cartilage.

## PHYSICAL EXAMINATION OF THE LIVER AND GALL-BLADDER

**Inspection.**—The method advocated by M. Knapp I have found most practical. The patient stands with the abdomen uncovered facing a good light, the examiner slightly to the patient's right, so as not to interfere with the exposure to light. The lower edge of the enlarged liver shows on the abdomen as a linear transverse shadow, moving up and down with respiration and being especially prominent at the close of expiration, when there is a sudden check to the movement. Pulsation can occasionally be observed. Extreme enlargement may cause fulness in the right hypochondrium.

An enlarged gall-bladder can be appreciated in the same manner. Inspection may also be made with the patient in the dorsal position, the examiner stooping so as to bring the eyes on a level with the abdomen.

**Palpation.**—The head and shoulders should be slightly raised and the knees and thighs flexed, the patient being in the dorsal position. The examiner, sitting on the right side of the patient, should lay the right hand flat on the abdomen below the right costal arch, the fingers pointing upward and obliquely inward just to the right of the right rectus. Depress the fingers and feel for the resistant edge of the liver. The patient should be directed to take deep respirations, and by pressing inward and upward with the fingers the edge of the organ can be felt to move up and down.

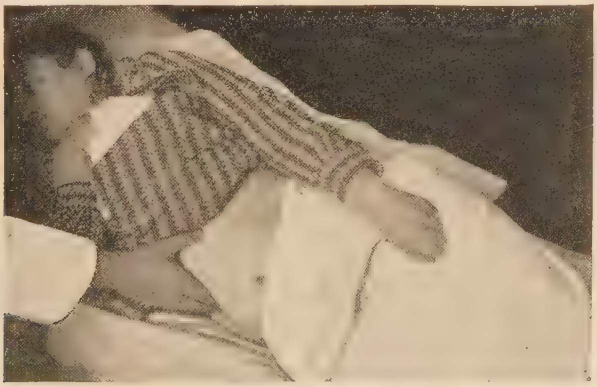

Fig. 35.—"Spilling" the liver.

As the liver may be enlarged, palpation for its lower edge must be begun from the level of the umbilicus upward. It is often necessary to feel for the notch of the gall-bladder or round ligament to determine whether it is the edge of the liver which is felt on palpation. One should note whether the edge is sharp or thick, or smooth or irregular. In some cases with thick abdominal walls the liver may be " spilled " against the latter by turning the patient on the right side, as in Fig. 35, thus rendering palpation more easy. The hand in this case may be in the reversed position.

The surface of the liver should be palpated, the left lobe in the epigastrium and the portion projecting below the ribs, if it be enlarged.

One should observe whether it is rough, smooth, nodular, or whether large tumor-like masses are present; also whether it is hard or soft and fluctuating; or if there is a thrill (hydatid), or friction during respiration, or pulsation. If the abdomen is distended the " dipping " method of palpation may be necessary.

The *empty gall-bladder* is not palpable. If distended it feels like a smooth pear-shaped tumor, moves with respiration, and is movable laterally, unless there are adhesions.

If malignant growth is present, the gall-bladder is irregular and nodular. If there are many gall-stones, Hutchinson describes the feel as of a " bag of nuts."

**Percussion of Liver and Gall-bladder.**—For anterior and lateral percussion the patient should lie down; for percussion posteriorly he should be sitting or standing.

The upper part of the right lobe is overlapped by the lung, and of the left lobe, a small area is covered by the left lung and heart.

Percussion over the covered part gives impaired pulmonary resonance, or modified dulness (deep, relative, or covered hepatic dulness). The part in contact with the parietes gives absolute dulness (superficial or exposed dulness).

It is necessary to delimit the entire area. Percuss downward, first in the mammillary line, beginning at the second intercostal space; then in the midaxillary line from the fourth interspace, and in the scapular line from the angle of the scapula. Percussion should then be made from below upward, in the midline from the umbilicus and from lateral and posterior points below the ribs.

*Covered Hepatic Dulness.*—Strong percussion should be employed, commencing above in the areas noted, and watching for the change from pure pulmonary resonance to impaired resonance, which denotes the presence of the liver. Impaired resonance is found normally in the fourth space in the mammillary line; in the seventh space in the midaxillary line; and in the ninth space in the scapular line.

*Upper Limit of Exposed (Absolute) Hepatic Dulness.*—Gentle percussion should then be employed and normally absolute liver dulness appears in the mammillary line at the sixth rib; midaxillary line at the eighth rib; and in the scapular line at the tenth rib.

*Lower Limit of Hepatic Dulness.*—Gentle percussion along the lines previously indicated from below upward will differentiate between tympanites and hepatic dulness. The lower limit normally is in the median line anteriorly a hand-breadth ($3\frac{1}{2}$ to 4 inches) below the ensiform; in the mammillary line, the tenth space. In the scapular line, it joins the dulness of the right kidney.

The vertical width of liver dulness is normally in the mammillary line 4 inches; in the midaxillary, 6 inches; in the scapular, 3 inches.

*Percussion of the Gall-bladder.*—This is only possible when it is distended or enlarged, in which event there is an area of dulness projecting downward and inward from the lower border of the liver and continuous with the dulness of the latter. In some cases the transverse colon may pass over the neck of the distended gall-bladder and separate its dulness from that of the liver by a tympanitic area. This is important to remember.

*Auscultatory Percussion of the Liver.*—The stethoscope should be placed over the middle of the area of the liver anteriorly, laterally, and posteriorly, and percussion be carried out on the lines already described. As a rule, simple percussion is sufficient; but the auscultatory method is of special value to determine whether a tumor is connected with the liver or not.

Thus, in Fig. 36 the stethoscope is placed over point *S*. The note over the tumor *T* resembles more closely in intensity and quality the percussion note over the liver at *C* than it does over the point *A*.

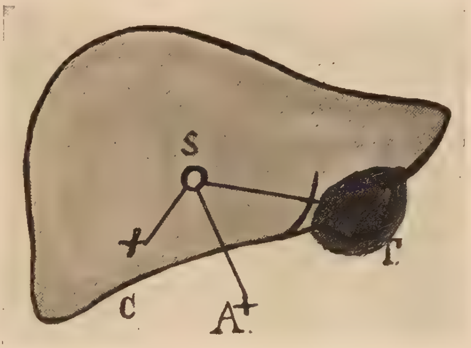

Fig. 36.—Tumor of liver.

General enlargement of the liver may be due to passive congestion, usually from valvular disease of the heart, amyloid disease, cancer, fatty infiltration, hypertrophic cirrhosis, leukemia, abscess, gumma, or, rarely, Weil's disease.

Circumscribed enlargement of the liver, *i. e.*, of the left lobe, is usually due to abscess, hydatid cyst, gumma, or cancer.

Downward displacement of the liver is caused by various intrathoracic lesions, such as emphysema, effusions, etc., by subphrenic abscess, or as a part of a general ptosis of the viscera. In downward displacement the upper surface, especially of the left lobe, is readily accessible to palpation and presents a rounded surface.

A prolapsed liver does not move as freely with respiration, on account of its separation from the diaphragm.

The consistence of the liver is somewhat diagnostic. It is abnormally hard, dense, and resistant in cirrhosis, carcinoma, amyloid disease, or syphilis.

A fluctuating swelling at the lower border may be a distended gall-bladder, abscess, or hydatid cyst.

The surface is smooth in fatty infiltration or degeneration, in passive congestion, and in amyloid disease; it is rough in tubercular peritonitis and granular to the feel in cirrhosis.

Hard nodules, varying in size, are suggestive of cancer; smooth, elevated prominences occur with gummata. A smooth projection may be due to abscess or cyst.

**Topography of the Pancreas.**—The pancreas (Fig. 37) lies about 3 inches above the umbilicus, midway between the navel and the ensiform appendix, corresponding to the level of the first lumbar vertebra.

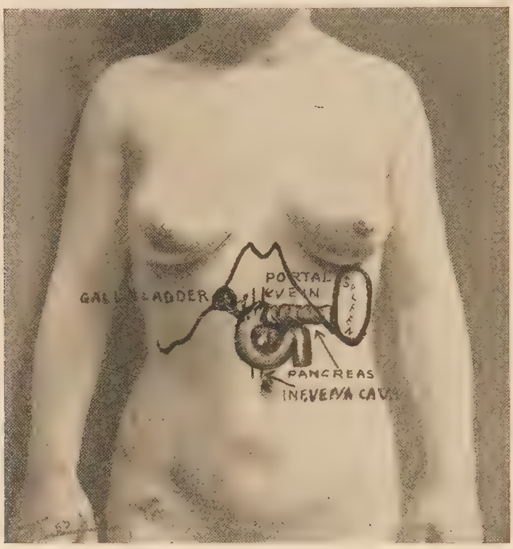

Fig. 37.—The pancreas.

It is about 6 inches long and lies deep in the epigastrium, transversely across the spine, with its head resting in the curve of the duodenum and its tail extending to the spleen. The stomach covers it in front. It is rarely accessible to direct examination. The head of the organ lies in close relation to the inferior vena cava, portal vein, and common bile-duct, which are posterior. A cancer or growth of the head of the pancreas may press upon these blood-vessels and cause edema and ascites, or upon the bile-duct and produce persistent jaundice with dilatation of the gall-bladder.

Pain, fatty diarrhea, ascites, glycosuria, and jaundice may result from pancreatic disease.

The diseases in which these conditions are present are acute hemorrhagic or suppurative pancreatitis, chronic pancreatitis, and tumor—either carcinomatous or cystic.

Under Diseases of the Pancreas the author refers his readers for a complete description of the subject.

*Physical Examination.*—Normally the pancreas cannot be palpated unless the patient is extremely emaciated.

An important physical sign of pancreatic disease is the *presence of a tumor* in the median portion of the epigastrium, midway between the navel and the ensiform process. It is deep seated and often nothmore *than a sense of resistance* can be appreciated by the palpating hand.

**Topography of the Spleen.**—The spleen is oval, flattened in shape, and lies in the left hypochondriac region, measuring on an average 5 by 3 inches. It reaches from a point 1½ inches from the midspinal line posteriorly nearly to the midaxillary line, lying along the ninth, tenth, and eleventh ribs, the long axis being parallel with the ribs and running obliquely forward and downward, as in Fig. 40.

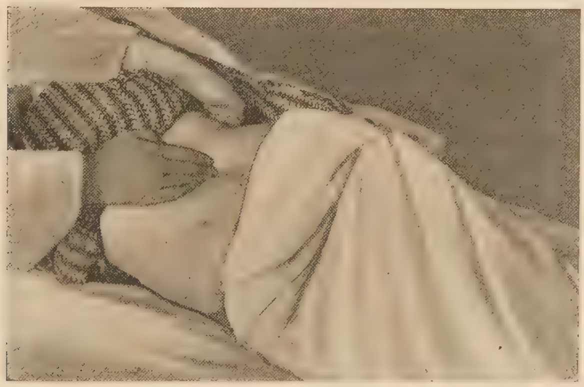

Fig. 38.—Examination of the spleen. "Spilling" the spleen.

The lower two-thirds of its outer surface lie against the lateral abdominal wall, and the upper third is overlapped by the diaphragm, which separates it from the lower border of the left lung. The diaphragm lies above and the left kidney posteriorly, and it is in contact elsewhere with the stomach, pancreas, colon, and small intestine.

The anterior border is sharp and indented by two to four notches.

**Physical Examination of the Spleen.**—*Inspection.*—If the spleen is greatly enlarged, it may be visible as a protuberance extending from the left hypochondrium downward and inward, moving with respiration.

*Palpation.*—With the patient in the dorsal position and the knees and thighs flexed, the examiner on the right side of the bed should lay the right hand flat on the abdomen and with the fingertips exert pressure, pushing obliquely upward under the left costal margin at the tenth cartilage. The edge of the enlarged spleen can, as a rule, then be felt.

If it is not palpable, then request the patient to take deep breaths, when the sharp edge of the organ, which is smooth and usually notched, moving with respiration, will be felt riding over the finger-tips and directed downward and inward.

The left hand may be placed posteriorly between the ends of the tenth and eleventh ribs and firm pressure be made, so as to tilt the organ forward and thus make palpation more easy. Normally the spleen cannot be felt.

If the organ is enlarged a depression or space, into which the finger-tips can be sunk, can be detected at the posterior border of the enlarged spleen between it and the erector spinæ.

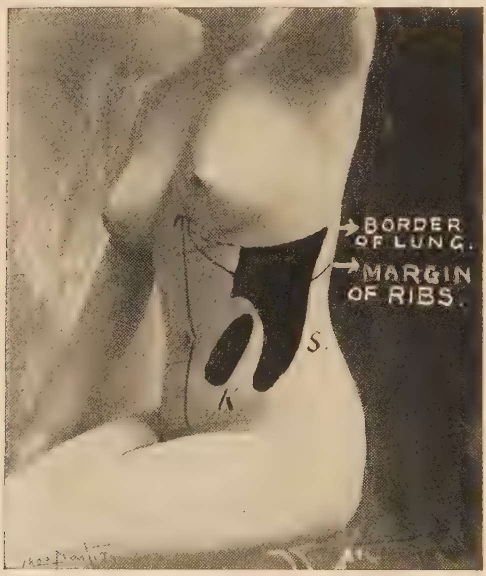

Fig. 39.—*S*, Tumor of spleen; *K*, tumor of kidney. Kidney tumor overlaid by tympanitic colon.

An excellent method of palpation of the spleen is shown in Fig. 38. By turning the patient on the right side, combined with posterior pressure, palpation is rendered easier.

It is often necessary to differentiate between tumor of the left kidney or spleen.

The spleen is oval, moves with respiration, is notched, has a sharp edge, a gap is present between it and the lumbar muscles, and it has no tympanitic resonance over it.

The kidney is reniform in shape and rounded, has no sharp edge or notch, and is overlaid by tympanitic resonance (Fig. 39).

*Percussion of the Spleen.*—The patient may be recumbent, partially turned to the right, midway between the dorsal and right lateral position, with the left arm extended over the head; or the examination may be made in the sitting posture. Percussion should be light, except over the posterior portion near the kidney.

It should be carried out along the lines *A*, *B*, *C*, *D* in Fig. 40. Anterior percussion at the costal margin along the tenth rib, along *A* until the tympanites of the stomach is replaced by dulness, usually at midaxillary line. From above percuss along *B*, commencing at the level of the angle of the scapula midway between the posterior axillary and scapular lines, passing vertically downward until pulmonary resonance is impaired, generally at ninth rib.

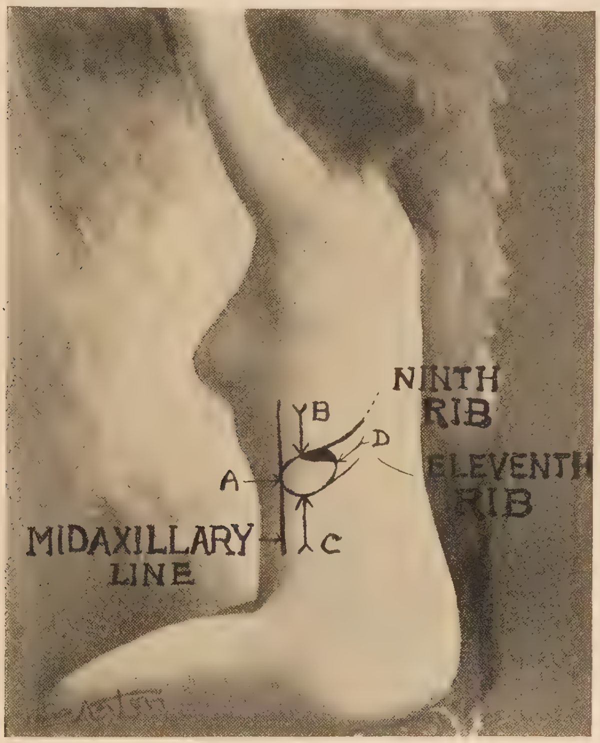

Fig. 40.—Lines for percussion of spleen.

Percuss from below upward along line *C*, commencing below the border of the ribs, in or slightly posterior to the posterior axillary line, and passing upward until tympanites become dull, usually at the eleventh rib.

Posteriorly percuss strongly from midspinal line at level of tenth rib and along the latter.

Splenic dulness should commence 1½ inches from the median spinal line. It is difficult to determine. The area of splenic dulness is oval, 2 to 2½ inches by 3 to 3½ inches. Dulness of over 3½ inches, on vertical percussion, shows enlargement.

Pleuritic effusion, consolidation at the left base, and fecal accumulation in the splenic flexure, may obscure percussion of the spleen. Palpation is the most important method and the most accurate.

Acute enlargement of the spleen occurs with infectious diseases, such as typhoid, malaria, etc., and in septic processes; chronic enlargement, with leukemia, malaria, cirrhosis of the liver, amyloid disease, pernicious anemia, etc. The organ may be displaced downward by intrathoracic pressure.

Abscess, carcinoma, or hydatids may cause an unequal enlargement. Liver and spleen may be enlarged together in passive congestion, cirrhosis of the liver, leukemia, and in amyloid disease.

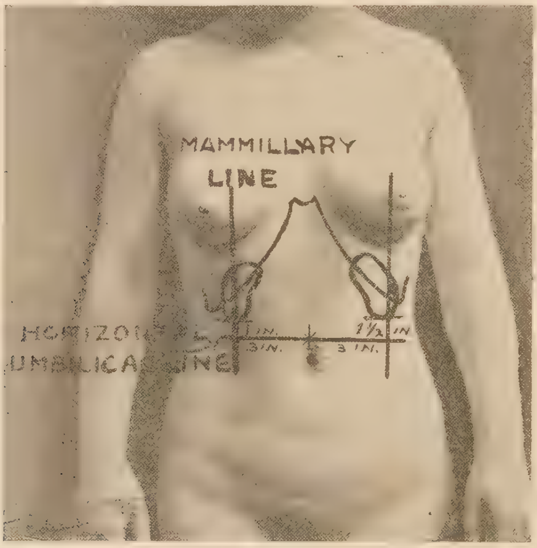

Fig. 41.—Anterior surface of kidneys.

A floating spleen may occur as a result of congenital laxity of its ligaments or to overstretching from the increased size or weight. It may be part of a visceroptosis, usually in women. It is recognized by its mobility, shape, sharp edge, and notches.

**Topography of the Kidneys.**—The two kidneys lie against the posterior abdominal wall, one on each side of the spinal column, in beds of fat and connective tissue. They are of reniform shape. The upper end of the right kidney is in contact with the liver, and the left kidney with the spleen. They are retroperitoneal, the ascending and descending colon respectively lying in relation in front. The right kidney lies about ½ inch lower than the left. Each organ is about 4 inches long, 2 to 2½ inches in breadth, and 1 inch thick.

*Surface Relations of the Kidneys.*—Draw a horizontal line through the upper margin of the umbilicus; prolong the mammillary lines on each side downward until they intersect this horizontal line. The points of intersection lie about 3 inches on each side of the median line.

From the intersections measure upward 1 inch on the right and 1½ inches on the left mammillary line, and draw on each side a short horizontal line. The lower ends of the kidneys lie at these levels (Fig. 41).

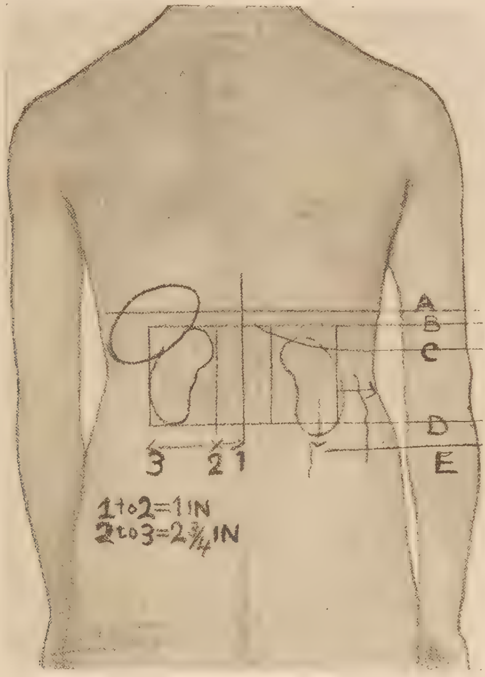

Fig. 42.—Posterior surface relations of kidneys: *A*, Lower border of lungs; *B*, level of spine of eleventh dorsal vertebra; *C*, lower border of liver; *D*, level of spine of third lumbar vertebra; *E*, colon.

One can measure 3 inches on each side along the horizontal line and then upward, without drawing the mammillary line. The kidneys extend upward and inward about 4 inches, one-third to the outer side and two-thirds to the inner side of the vertical lines.

*Posterior Surface Relations of the Kidneys.*—Draw a horizontal line across the back at the level of the tip of the spine of the eleventh dorsal vertebra; a second line at a level of the tip of the spine of the third lumbar vertebra. On each side draw a vertical line from the upper to the lower horizontal lines, 1 inch from the median line of the spine, and second vertical lines 2¾ inches away from the first vertical lines. Within these outer parallelograms lie the kidneys (Fig. 42).

The lower ends of these organs lie from 1 to 1½ inches above the iliac crests, the right ½ inch lower than the left. About a third of the upper ends are covered by the eleventh and twelfth ribs; the liver overlaps the right kidney and the spleen the left.

**Physical Examination of the Kidneys.**—Inspection is seldom of service; palpation is most valuable; percussion is often uncertain.

*Inspection.*—A large tumor of the kidneys may be visible in the anterior lumbar regions, extending into the umbilical region, with outward bulging of the ribs on the affected side, such as in the case of sarcoma, hydronephrosis, or cyst. A perinephritic abscess may become visible as a swelling in the posterior lumbar region.

*Palpation.*—This is most important. There are several methods described, of which the two following are the most practical:

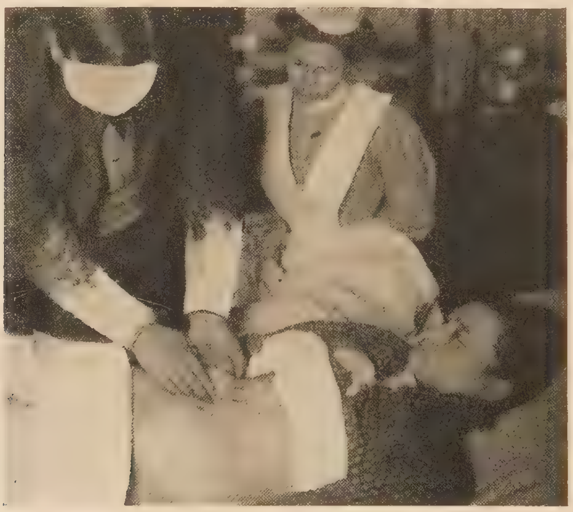

Fig. 43.—Palpation of kidney (older method).

*Method 1.*—The patient lies in the dorsal position, with the knees and thighs flexed, and the head is slightly raised to secure perfect relaxation. If the right kidney is to be examined, the left hand is slipped under the back, so that it rests on the two lower ribs and the lumbar space below them. The right hand is laid flat on the abdomen in front, resting below the costal margin to the outer side of the rectus, as in Fig. 43.

The patient should take deep and slow respirations, and during expiration firm pressure should be made with the fingers in front against posterior counterpressure, so that the kidney may be grasped between the hands.

If the kidney is normal in position and size, the extreme lower edge can be felt if the abdominal walls are not too thick. If the lower quadrant of the organ can be clearly palpated, it may be considered a movable kidney (of the first degree).

Various classifications of mobility have been given, some considering the organ when palpable in half its extent, mobility of second degree; entirely palpable, mobility of third degree; and when descending into the abdominal cavity, mobility of fourth degree. Others consider it movable if the entire length be accessible, especially if it can slip down as far as the horizontal umbilical line; and if it can be displaced below this or across the abdomen, as a floating kidney.

I believe that any kidney which can be palpated to one-fourth of its extent should be considered movable, and the subdivision into various degrees of mobility to be excellent. A movable kidney from strain or traumatism is comparatively rare. Occasionally we find a congenital floating kidney with no visceroptosis. As a rule, I believe that in about 95 per cent. of cases of movable kidney the condition is merely part of a ptosis of the abdominal organs, and it may be considered to be practically pathognomonic of this condition. The right kidney is most frequently movable, though both may be so.

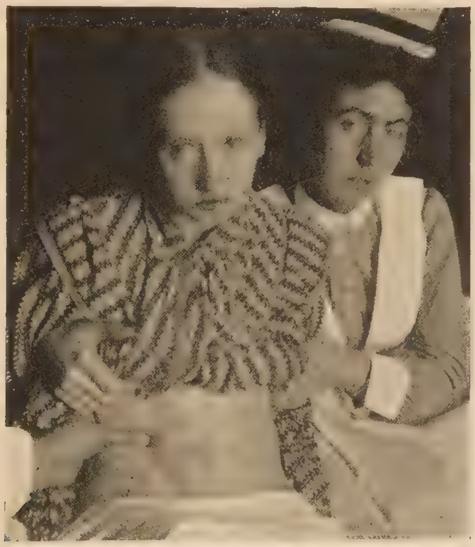

Fig. 44.—Palpation of kidney. Step 1.

If the kidney is not found at its normal site, it should be searched for in the abdomen.

In examination of the left kidney, the position of the hands is reversed.

*Method 2.*—This is, from personal experience, the most practical method of palpation, and the varying degrees of mobility can be detected with greater ease than by the method already described.

For examination of the right kidney, the patient sits up in bed, and the left hand of the examiner grasps the right flank, the thumb resting under the costal margin, the fingers posteriorly. The patient breathes deeply, or coughs, or bears down, and the kidney can be felt to slip down between the thumb and fingers, like "a pea in a pod," or the lower margin or part of the kidney may be felt (Fig. 44).

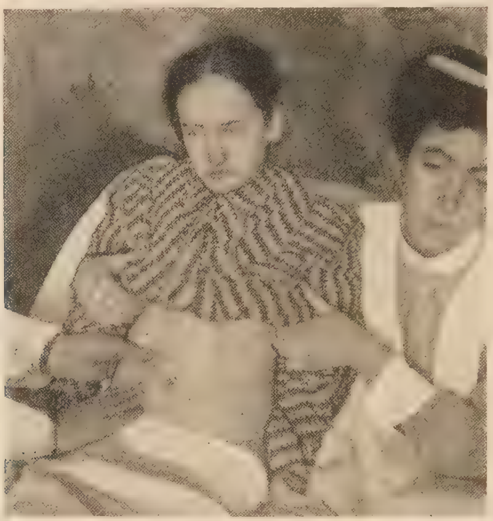

Fig. 45.—Palpation of kidney. Step 2. Patient in semi-oblique posture.

If the organ slips beyond the thumb and fingers or is not found in its normal position, then the left hand grasps the flank more firmly and upward palpation is made with the right hand, as in Fig. 45, the patient gradually assuming the semi-oblique and, finally, the dorsal position, the final step, as shown in Fig. 46.

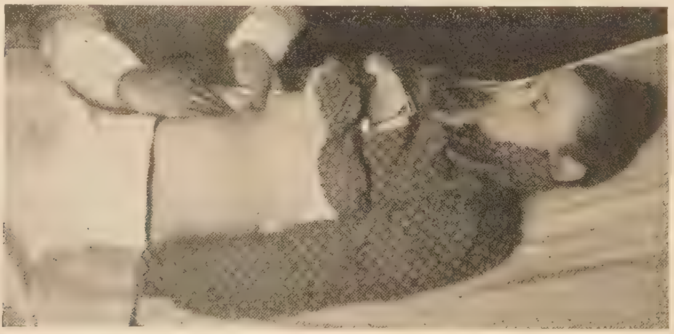

Fig. 46.—Palpation of kidney. Step 3 (final). Patient in dorsal position.

A similar method with the patient standing is employed by Goelet, but the technic described seems preferable.

For palpation of the left kidney, the position of the hands is reversed, the right hand grasping the loin and the left hand anteriorly.

Tumors, hydronephrosis, and cysts of the kidney may be detected by palpation of the abdomen, as already described.

*Brewer's Point.*—Tenderness at the costovertebral angle is diagnostic of an acute inflammatory condition of the kidney, such as infarctions, etc., and aids in differentiation between this condition, appendix adherent to the liver, and acute cholecystitis. A small area of tenderness may be found overlying the lowest two or three ribs[1] when that at Brewer's point is absent.

*Percussion of the Kidney.*—The lower and part of the outer border of the kidney may at times be determined by percussion, comparing the dulness with the tympanitic note of the colon which lies anteriorly. As a rule, the thickness of the muscles, peritoneal fat, or fecal accumulation renders the results uncertain. Inflation of the colon with air or carbonic acid gas may aid in outlining renal dulness or percussion by comparison with the tympanitic area. A clear tympanitic note will be given if the kidney is absent (prolapsed) on that side.

*Position for Percussion.*—The patient may lie on the abdomen, with one or two pillows placed underneath, to arch the back; or on the side, midway between the lateral and prone position, the physician sitting facing the patient's back and percussing the uppermost kidney.

One should commence percussion in the middle of the area in which the kidney lies (Fig. 42), using heavy strokes, and percuss outward until the kidney dulness is replaced by tympanites; also percuss downward in the same way. Increased dulness shows enlarged kidney. Lerche[2] holds that, with percussion hammer and ivory plate (plexor-pleximeter), and using a drop *stroke* (not tapping), better results are secured.

*To Differentiate a Movable Right Kidney from the Distended Gall-bladder.*—The kidney is movable in all directions, can be carried downward, does not move with respiration. Tympanites is found between it and the costal margin; the shape is reniform, and it may be pushed back into its normal position.

The distended gall-bladder moves with respiration, and can only be moved laterally; if pushed away from the abdominal wall, it tends to resume its original position, and there is no tympanites between it and the liver dulness.

The chief causes of enlarged kidney are pyonephrosis, perinephritic abscess, hydronephrosis, cysts, carcinoma, and sarcoma.

An enlarged kidney tends to develop toward the front; an abscess, posteriorly, between the last rib and the iliac crest.

With enlarged kidney, the colon overlays in front of the tumor. With splenic tumor, this does not occur (Fig. 39).

If in doubt, inflate the descending colon with air through a colon-tube or catheter.

[1] Med. Rec., July 15, 1911.

[2] Ibid., Feb. 4, 1911.

# PART II

# DISEASES OF THE STOMACH

## CHAPTER V

## METHODS OF PHYSICAL EXAMINATION OF THE STOMACH

**General Considerations.**—For an intelligent understanding of the subject, it is necessary briefly to define those conditions which constitute an abnormality in the position of the stomach and to differentiate between them.

The greater curvature of the normally distended stomach lies about 2 to 3 fingers-breadth (1½–2¼ inches) above the umbilicus. The normal position of the organ has been indicated in Chapter I. It must be remembered that some possess an abnormally large stomach, and that it can be considered to be dilated only if there are symptoms associated which point to this organ.

If there is atony of the stomach, with motor insufficiency, the patient having gastric symptoms, while the *lower border of the stomach is defined* at the level of the umbilicus or below it, the lesser curvature maintaining its relations to the diaphragm, we may consider the organ to be dilated. This constitutes the atonic type of dilatation, which is extremely common. Many of this class suffer from auto-intoxication with nervous sequelæ, and are found in great numbers in our asylums and among our nervous cases. Attention may be diverted from the gastric symptoms and they may even be slight.

As a result of pyloric spasm, or benign or malignant stricture of the pylorus, or any obstruction in the pyloric region interfering with the egress of the gastric contents, we have the so-called stenotic type of dilatation of the stomach.

In these cases the lesser curvature retains its relation to the diaphragm, while the lower border extends to the umbilicus or below it, and gastric symptoms are present to a marked degree and of a special type.

With gastroptosis (prolapse of the stomach), the suspensory ligaments of the stomach are relaxed and the entire organ sinks, *the lesser curvature as well as the greater.* In aggravated cases the lesser curvature looks inward to the right, and the greater curvature outward to the left. The pylorus may often lie below the level of the umbilicus. With this condition, movable kidney—especially of the right organ—is present, and may practically be considered pathognomonic, in my opinion, as only in about 5 per cent. or less of cases is mobility due to strain or injury. The congenital floating kidney, without ptosis of other viscera, is exceedingly rare.

There may be various degrees of gastroptosis, a loop-shape, or even a vertical stomach, similar to that of the fetal period, which are pictured in the chapter on Gastroptosis. *The change in position of the lesser curvature is*, therefore, *diagnostic of gastroptosis*, and not the position of the lower border of the stomach. *The presence of movable kidney is also diagnostic.*

It seems advisable to describe the methods generally employed, and at the end of the chapter briefly to summarize those which are of most practical value.

**Preparation of the Patient.**—On the day or night previous to examination the bowels should, if possible, be thoroughly emptied by a cathartic. If there is much tympanites, it should be relieved by a hot enema of about 1 liter (1 quart) of normal saline solution or soapsuds and water; or, if the condition is marked, then by enteroclysis at 110° to 120° F. with the recurrent tube (Kemp's) or two catheters. This carries off the gas in a satisfactory manner.

The patient should be examined in the dorsal, semi-oblique, and standing positions.

**Inspection.**—Examination in the dorsal position should first be made.

A recognizable bulging, distinct from the epigastrium, especially if it occur in the umbilical or hypogastric region, may be due to a dilated stomach; the epigastrium, under these conditions, is usually hollow and depressed. Inspection is often of assistance in thin patients, especially after artificial distention of the stomach with carbonic acid gas.

Peristaltic movements of the dilated stomach are at times observed.

Kussmaul has noted very active peristaltic movements in the dilated stomach (peristaltic unrest), the waves passing from the linea alba below the umbilicus in an upward direction and to the right to the lower margin of the liver. This is found present in cases in which stenosis of the pylorus exists.

We can facilitate inspection by placing the patient upon a raised table, the head toward the window, the shades being arranged so that the light enters on a plane only slightly above that of the patient, and is directed from the head toward the feet. The examiner, standing toward the foot of the table and bending from side to side, can at times make out shadows cast by the inequalities of the abdomen. The shadows move with respiration. By this method the size, shape, and position of the stomach can often be made out.

Knapp places the patient in the same position, but stands at the side or at the shoulders, and brings his eyes down to the level of the abdomen and observes the respiratory waves passing over its surface. After some experience one can detect delicate transverse lines or waves passing upward and downward with respiration. These lines correspond to the curvatures of the stomach.

More recently he places the patient facing a good light, and, standing slightly to the side, observes the movements of the transverse lines. I have seen good results from this method, especially for determination of the lower border of the stomach.

The following signs I have found quite reliable: With the patient in the recumbent position, a marked concavity between the costal arches—extending from the ensiform process to or below the umbilicus, with a vertical median sulcus, wider above than below, the abdomen being flattened in the central part and bulging in the lateral regions—is significant of gastroptosis. In the erect position the epigastrium becomes still more depressed, while the umbilical and especially the pubic regions bulge outward. Tumors of the stomach may sometimes be observed, causing slight projection or protuberance on the abdominal wall.

**Palpation of the Stomach.**—Inspection should be supplemented by palpation. Palpation should be performed gently, and the hands of the operator should be warm.

The patient should be in the dorsal position, with the legs flexed, to relax the abdominal muscles. He should breathe naturally and keep the mouth open to aid relaxation. The physician should be seated on the right side of the bed and palpate with the right hand, which should be flat or slightly bent upon the abdomen, with the ulnar side down. One can stroke from above downward, and with practice it is possible, in some cases, to feel the stomach-wall and appreciate the position of the greater curvature, as the stomach gives a more uniform elastic sensation than do the intestinal walls. Some commence palpation from below and work upward, dipping in the ulnar edge of the hand rather deeply. By these means it is at times possible to determine the position of the greater curvature.

By palpation we can discover if nephroptosis is present. Diastasis of the recti muscles and floating tenth rib can also be determined. These conditions are significant of gastroptosis. Under Inspection I have noted the signs that are significant of gastroptosis. If we find a " movable kidney," this renders our diagnosis conclusive.

By gentle palpation one can frequently discover a tumor, its position, size, consistency, and mobility. Occasionally, more pressure is necessary, and the palpating hand may be reinforced by the other hand, after the method described. (See Fig. 27.)

Sensitive or tender points can be located by palpation; for example, the circumscribed tenderness of an ulcer or the diffuse tenderness of the gastric region in acute inflammation. Boas has devised an algesimeter for indicating the degree of pain. T. Kilmer has also an instrument for the same purpose.

Considerable care should be exercised in palpation in cases of suspected ulcer, and I prefer the hand for this purpose.

**Percussion of the Stomach.**—The accurate determination of the position and size of the stomach is often difficult by simple percussion. The sound varies, according to whether the organ is empty or filled with air, food, and water.

The position of the patient, whether lying down, semi-oblique, or standing, modifies the findings. In order to obtain results the stomach should contain some air. Dehio has demonstrated, both on living subjects and on the cadaver, that *if the stomach is empty*, the tympanitic sound which we produce on percussion is due to the colon and not to the stomach, since the latter is contracted into the left concavity of the diaphragm and is not in contact with the anterior thoracic wall. Hence the time at which the examination is made is important. Moreover, the *lower curvature* tends to fall away from the abdominal wall.

The patient should first be examined in the dorsal position with the knees flexed.

This method determines with fair accuracy the upper right and upper left portions. The percussion hammer is sometimes an aid. The absolute determination of the lower border by percussion is more difficult. It is rendered easier if the bowels have been thoroughly emptied, since the colon is then less likely to ride over the greater curvature. The percussion sound over the colon is lighter and does not equal that over the stomach. The stomach sound is of greater intensity and clearness and of higher pitch. This, of course, refers to conditions when air is present as the factor. Food or fecal contents alter the result, which is further modified by percussion in the semi-oblique and standing positions.

As a rule, there are some contents in the transverse colon, so that we have the tympanites of the stomach merging into dulness or flatness. With gastroptosis, determination of the position of the organ by simple percussion is often difficult.

The presence of a tumor can frequently be determined by percussion.

*Dorsal Gastric Nucleus of Resonance.*—William Ewart, in his latest communication in the Proceedings of the Royal Society of Medicine, July, 1910, has contributed a new method of post-gastric percussion, and has described a hitherto unrecognized "dorsal gastric nucleus of resonance." This consists of a circular area, from 2 to 2½ inches in diameter, situated immediately below the inferior angle of the left scapula. Over this area the percussion-note is one of increased resonance and of a tympanitic quality. This is the result of the deep-seated resonance of the stomach, the constancy of its position and its circular shape being determined by sound refraction, the liver acting as a lens for the resonant waves. The clinical value of this sign is concerned chiefly with the diagnosis of the various forms of dilatation of the stomach. The loss of perfectly circular outline is the first step toward the disappearance of the gastric nucleus, which may be the starting-point of progressive encroachment outward of an enlarging area of dorsal gastric resonance. This is the dorsal or backward type of upward dilatation of the stomach which was first definitely described by Ewart, who believes this to be the cause of one of the most severe and dangerous forms of heart distress of mechanical gastric origin.

**Auscultatory Percussion.**—With this method we employ the stethoscope. The chest-piece may be placed above the seventh rib in the left mammillary line, or between the tip of the ensiform process and the left costal margin; or in the same vertical line, but slightly below these points. First percuss near the stethoscope to fix the characteristic sound. The tympanites of the stomach is transmitted generally through the liver and lung. The percussion should be begun *well distant* from the possible location of the stomach, and should be performed in the vertical direction, downward, upward, and also laterally. One should begin nearly at the symphysis and percuss in vertical lines upward (Fig. 47).

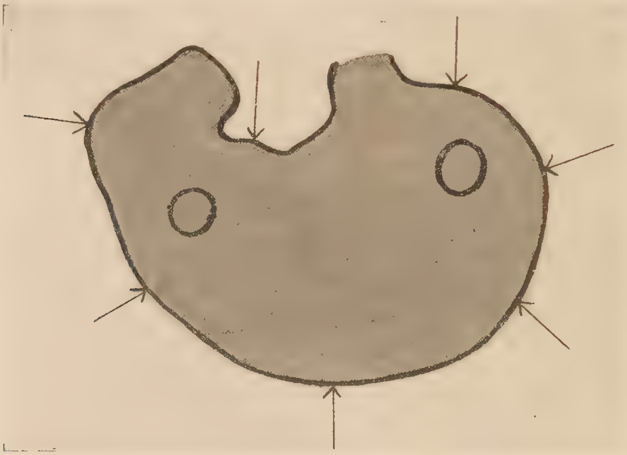

Fig. 47.—Vertical lines of auscultatory percussion. Circles show positions of stethoscope.

The patient should be in the usual position, as described, and should hold the stethoscope for the operator against the abdomen. A sound of greater intensity and clearness and of higher pitch denotes the border of the stomach. The greater bulk of the organ, when dilated or in a condition of ptosis, lies to the left of the median line.

We must remember that some cases of marked dilatation extend a great distance to the right of the abdomen.

This method is of value in determination of a tumor of the stomach.

Place the stethoscope at *O*, Fig. 48, and percuss toward the tumor from all directions. The sound heard over the tumor (*C*) differs in character from that heard over the stomach at *D*. If the growth involves the stomach wall, *C* resembles *D* much more nearly than *A* (percussion of liver) and B (percussion of intestines) resembles *D* (stomach percussion).

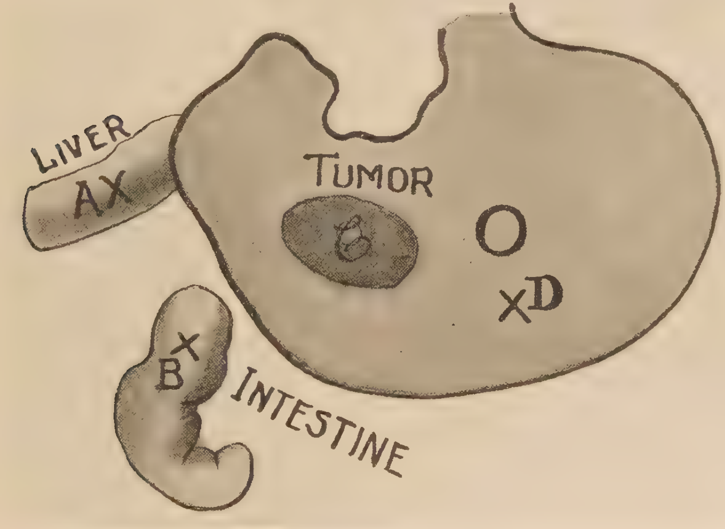

Fig. 48.—Auscultatory percussion of tumor of stomach-wall.

**Differential Diagnosis.**—If a tumor lies at *D*, near the margin of the liver (Fig. 49), tumor of the liver is excluded as follows: place stethoscope at *S*, over liver. Percussion note over *D* resembles note over *G* more than it resembles that over *F*. Then to *exclude intestines*, shift stethoscope to *K*, over stomach. Percussion over *D* resembles that over *M* more closely than that over *G* resembles that over *M*. The tumor is, therefore, of the stomach.

**Reichmann's Rod.**—This consists of a short ivory rod, with circular grooves and intervening projections, like the handle of an ivory knitting-needle. The rod is pushed firmly down over the stomach at a right angle to its surface (in a vertical line to the abdomen), and is gently stroked with the finger. The stethoscope is applied over the organ and the " pitch " carefully observed. When the rod passes beyond the limits of the stomach, a change in " pitch " occurs.

**Scratch Method of Auscultatory Percussion.**—This method I have found satisfactory in many cases.

The stethoscope is placed on the abdomen below the left border of the ribs, and with the index-finger of the right hand the abdominal wall is scratched gently by the examiner so as to secure the "normal pitch" over the stomach. The abdomen is then scratched lightly from below upward, commencing below the umbilicus, and the *change in "pitch"* is readily observed when the *lower border of the stomach is reached.*

If gastroptosis is believed to exist, the stethoscope is placed more to the left, and the "scratch pitch" noted from left to right. The method is shown in Fig. 50. It is of special value in determining the lower border of the stomach.

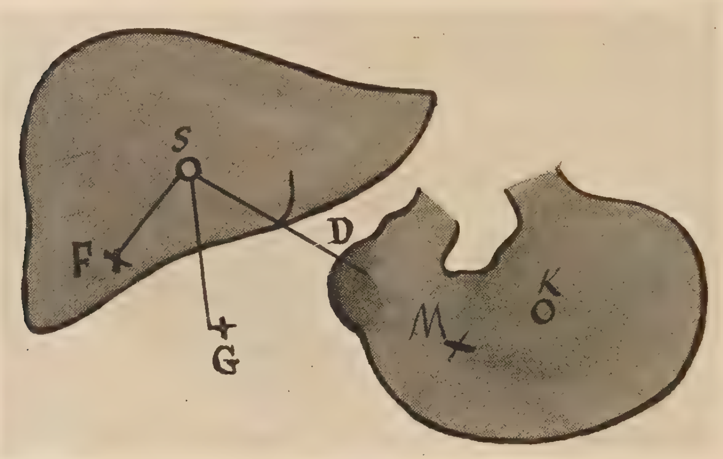

Fig. 49.—Differential diagnosis.

**Flicking.**—This method was described and illustrated in the previous chapter.

**Inflation of the Stomach with Carbonic Acid Gas.**—Carbonic acid gas inflation is employed to render the stomach visible to inspection; to aid the determination of the position of the lesser curvature, as well as the greater, and so enable a differential diagnosis between dilatation and gastroptosis.

The method is to administer, first, one-half glass of water in which about 1 dram of tartaric acid is dissolved, and, after this, one-half glass of water containing from 1 to 1½ drams of soda bicarbonate. If small quantities are employed, the stomach will not become visible and palpable.

There are certain objections to this procedure. At times there is considerable escape of gas through the cardiac orifice or pylorus and the small intestine may be distended. This is a possible source of error. There may be sudden hyperdistention of the stomach, with resulting pressure on the heart and lungs, and unpleasant or even dangerous symptoms result in the aged or in a patient suffering from cardiac or pulmonary disease. In the chapter on Acute Dilatation of the Stomach the effects of sudden distention of the stomach on the heart and circulation are described by Thomas Satterthwaite and the author.

When there has been a hemorrhage, or symptoms of ulcer or cancer, or signs of peritonitis, the use of this method is contraindicated. Several fatal accidents have occurred. It sometimes irritates the mucous membrane. One could employ a stomach-tube and Dr. Rose's carbonic acid gas generating-bottle as a substitute. Carbonic acid gas inflation is valuable in those who are in fair physical condition.

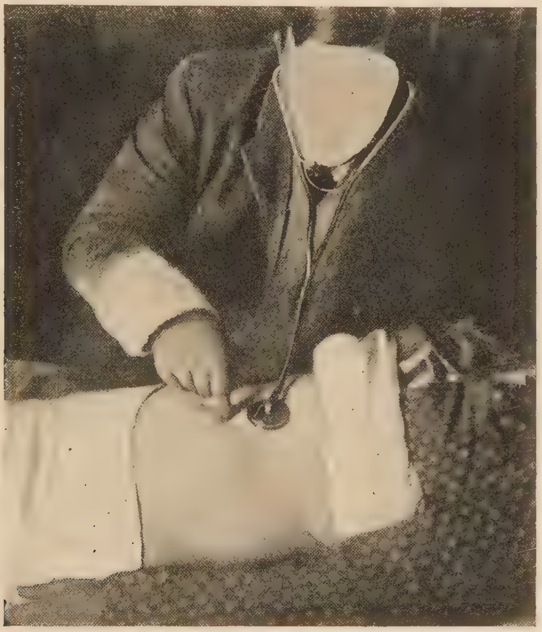

Fig. 50.—" Scratch method " of auscultatory percussion.

In the determination of the position of a tumor, whether it lies on the anterior or posterior surface of the stomach, it is of service. A posterior tumor will disappear under inflation. This is fully described under Cancer of the Stomach.

**Inflation of the Stomach with Air.**—This consists in introducing a soft stomach-tube and slowly pumping air into the stomach with a double-bulb or a Davidson syringe. The tube should be introduced with the patient sitting up in bed, and he should then gently recline on the back and inflation should be carried out. It possesses the advantage that the amount of air pumped into the stomach can definitely be regulated. Fill a vessel with 1 liter (1 quart) of water, invert it over a pail of water, and note how many compressions of the bulb displace the given quantity of fluid. One can thus estimate the quantity of air pumped in at each compression. The first few squeezes of the bulb should be given rapidly, so as to cause spasmodic closure of the pylorus.

The same indications and contraindications exist as for the use of carbonic acid gas. There is the advantage of being able to regulate more definitely the degree of inflation. Some patients, however, object to the passage of the tube. If there are discomfort or unpleasant symptoms from either method of inflation, the condition should be immediately relieved by the passage of the stomach-tube.

Fürbringer suggests that when we inflate with air the tube should be introduced only to the middle of the esophagus, and air should then be pumped in. He claims that this procedure prevents retching.

**Inflation of the Stomach with Water.**—To Dehio we must give the credit of determining the position of the stomach by water inflation. He percusses over the patient's stomach, preferably with the organ empty and the patient in the erect position. He then administers a glass of water (8 ounces), not too cold, and percusses the area of the dulness. He follows this with a second, third, and fourth glass of water, percussing each time, and notes the position and extent of the dulness. The patient is then directed to lie on his back, and tympanites will appear where dulness previously existed. This conclusively demonstrates that the area corresponded to the stomach.

If there is pronounced dilatation or ptosis, a single glass of water will often cause dulness to appear below the navel or in the inguinal region. The results may be obscured in patients with much adipose tissue or if there is fecal accumulation in the colon. In this event, it should be cleared out by injection. I have also found the following method of value, especially if there be some gastric contents: first, place the patient in the *semi-oblique position* and percuss the stomach; then administer 2 or even 3 glasses of water. We secure stomach tympanites above, then a band of stomach dulness, and intestinal tympanites below. It is easier to differentiate between dulness and tympanites than between two types of tympanites.

There are numerous complicated methods by means of inflatable bags, manometers, etc., for determining the position of the stomach which are scarcely of practical value. Leube introduces a stiff sound and determines the position of its lower end through the abdominal walls. This method does not seem to be safe. Others differentiate between the stomach and the colon by inflating the colon with air or carbonic acid, employing the same methods as in the stomach, only using twice the quantity of soda bicarbonate and tartaric acid. Rose's apparatus would prove of value to inflate the bowel. To further differentiate, water was given by the stomach. Some first empty the bowel thoroughly and then inflate the intestine with water. It is often difficult for the patient to hold the enema.

There are two other methods for determining the lower margin of the stomach: First, the administration of small quantities of soda bicarbonate and tartaric acid, with the patient in the standing position. In some cases one can approximately map out the lower border of the stomach by listening to the "sizzling sounds" with the stethoscope. Second, the use of the stomach-whistle (Fig. 51). This consists of a rectal tube of small caliber, with a whistle in the end. To the other extremity is attached an ordinary stomach aspirating bulb without valves. The tube is inserted into the stomach, the finger placed over the open end of the bulb, and a single bulbful of air is forced into and aspirated out of the stomach by rapid and short intermittent contractions. This entirely eliminates the possible chance of distending the stomach with air, and the organ remains practically empty.

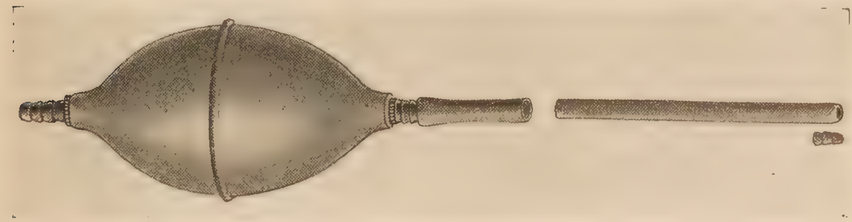

Fig. 51.—Kemp's stomach-whistle.

A stethoscope is placed over the abdomen and the point of greatest intensity of sound is marked by a cross with a colored pencil. The tube is pushed in and out and the various points of sound are marked—the lowest is in the lower border of the stomach. The ear can be applied in place of the stethoscope. Transillumination of the organ was then performed and the lower margins absolutely corresponded. The method of administering water and then blowing air into the stomach through a tube, and producing "bubbling sounds," only gives the level of the fluid, and not accurately the lower margin of the stomach. The whistle will not differentiate between dilatation and gastroptosis. This experiment with the stomach-whistle demonstrated that in the standing position the stomach, when empty, descends to the full length of its suspensory ligaments, and its lower border is at a constant level or within about 1 inch of the same, whether the organ be full or empty. It was at the same level when a pint or more of fluorescent fluid had been ingested as when demonstrated by transillumination. The stomach-whistle was described as early as 1892 by Spivak, of Denver, for the purpose of measuring the esophagus, and the author has been informed that S.

J. Meltzer had previously experimented with an instrument devised on the same principle. The writer does *not recommend* the whistle, but merely recounts its use as a matter of historic interest.

**Splashing Sounds.**—The splashing sounds of the stomach are produced when water and air in the organ are agitated together, when either the whole body or the stomach alone is shaken. They are best demonstrated by rapidly tapping with the index- and middle fingers of the right hand over the stomach several times in succession without removing the fingers, as in striking chords on the piano. The patient should be in the dorsal position, with the lower limbs flexed.

The sounds resemble those produced by shaking a rubber bag containing air and water.

They can be elicited in many people in ordinary good health shortly after meals, but if found at an abnormal time or in an abnormal position, are of diagnostic value. If present an hour after a test-breakfast, the patient suffering from gastric symptoms and the position of the stomach being normal, they are significant of simple atony. This is true if they be found several hours after an ordinary meal or on an empty stomach. If the splash is present in an abnormal position at the level or below the umbilicus, it shows the lower border of the stomach lies abnormally low, and that either dilatation or gastroptosis is present.

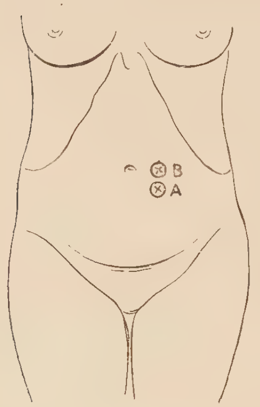

Fig. 52.—Splashing sound. Differential diagnosis between stomach and intestinal splash.

The presence of movable kidney demonstrates it is a ptosis. The upper border may be determined to be in an abnormal position by inflation or gastrodiaphany, as a further test.

The splashing sound determines the position of the lower border of the stomach with greater accuracy than percussion. Some patients hold the abdomen rigid, so that it does not appear on examination, but it can be produced artificially for examination purposes.

The following is a simple method to differentiate between stomach and intestinal splash:

In Fig. 52, if the splash be found at *A*, mark same on the abdominal wall; then give several glasses of water or, preferably, Vichy. If the splash at *A* be intensified, it is stomach splash. If, on the other hand, the splash appear at B, this is the *true* stomach splash and *A* is the intestinal splash.

If no splash is present on examination, it can be created artificially by giving several glasses of water or, preferably, Vichy. Sometimes 3 or 4 glasses are required; the patient takes several deep breaths, the splash being determined during expiration. I find the determination of the lower border of the stomach most accurate by means of the splashing sound.

**Deglutition Sounds.**—These were first described by Kronicker and Meltzer.[1]

When drinking, a sound is heard simultaneously with the act of deglutition, which is termed the first deglutition sound. A second sound is noted about seven seconds later. Both sounds can be heard by placing the ear or stethoscope at the ensiform process. As a rule, only the second sound is heard. If the first sound is heard, the second may be present or absent. The presence of these sounds assists in forming judgment as to the permeability of the cardiac orifice. If they are absent, the ingested liquid has remained in the esophagus, and hence a tight stricture is present. If the second sound is markedly delayed, there is probably partial obstruction.

**Other Sounds Have Been Described.**—Dripping sounds, arising from the passage of fluid along the gastric wall, are suggested as a means of mapping out the stomach. The method is inaccurate.

The succussion sound, obtained by shaking the body of the patient, is not as accurate a method as by tapping.

Gurgling sounds occur from the contraction of the empty stomach about air or gas. Sounds are heard in the stomach due to movements imparted to the organ through the respiration, and also ringing sounds, imparted from the heart in gastric dilatation.[2]

These sounds are of no diagnostic value. Occasionally sizzling sounds are heard on auscultation, which are produced by fermentation of the gastric contents. They resemble the sounds produced after the administration of soda bicarbonate and tartaric acid, with the resulting generation of carbonic acid gas.

**Esophagoscopy.**—Mikulicz, Rosenheim, Kelling, von Hacker,[3] Einhorn,[4] and many others have advocated this method.

In the earlier instruments the source of illumination was outside the esophageal tube. The instrument with the lamp at the end of the tube near the point to be inspected, such as devised by Einhorn, is more practical.

In Fig. 53 is depicted his instrument, which is readily understood.

The obturator is inserted and held in place by the plug *d* and the wires connected with the battery.

After introduction, the plug is removed, the obturator withdrawn, and the current turned on.

*Technic.*—The pharynx should be sprayed with cocain (5 per cent.). The patient sits on a chair with a straight back. The instrument with obturator inserted is immersed in warm water and inserted like a pen along the roof of the mouth to the posterior wall of the pharynx, the head being thrown backward on the shoulders, but not in too forced extension. The tube is then pushed down in a vertical line into the esophagus without the exertion of any force.

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[1] Centralbl. f. die med. Wissensch., 1883, No. 1.

[2] Laker, Wiener med. Presse, 1889, Nos. 43 and 44.

[3] Beiträge zur klinischen Chir., Bd. 20, 1898, pp. 141, 275; Ibid., Bd. 29, 1901, p. 128.

[4] New York Med. Jour., Dec. 11, 1897.

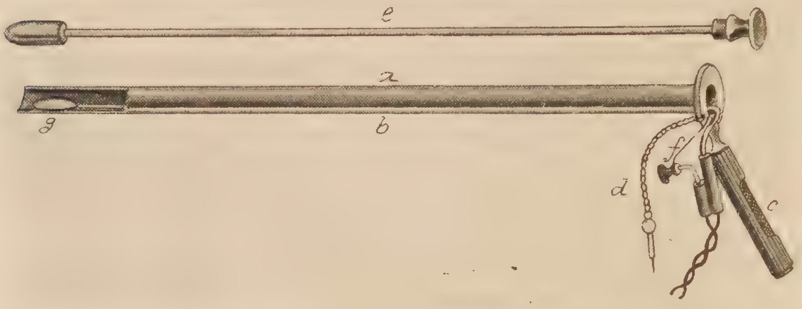

Fig. 53.—Einhorn's esophagoscope.

By this method it is unnecessary to press down on the tongue, and thus gagging is avoided.

The lips of the patient should not be pressed upon by the instrument. The obturator is removed, the light turned on, and the eye of the operator applied to the opening.

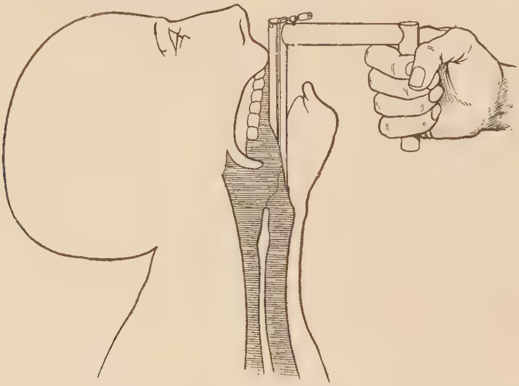

Fig. 54.—Misleading schema of direct bronchoscopy. Same position improper in esophagoscopy (Chevalier Jackson).

For complete inspection of the esophagus, the instrument is gradually withdrawn.

This esophagoscope is made in several lengths and in two sizes.

Chevalier Jackson[1] has devised an improved esophagoscope which contains an auxiliary tube for drainage and suction of the secretions. For esophagoscopy frequently the upright position can be employed, though in some cases the dorsal position is preferable. Fig. 54 is the incorrect position. In Fig. 55 Jackson shows the correct position of the head when bronchoscopy is performed in the upright position. The posture is the same in esophagoscopy. In Figs. 56 and 57 are shown the correct and incorrect positions for esophagoscopy in the dorsal position.

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[1] Jour. Amer. Med. Assoc., Sept. 25, 1909.

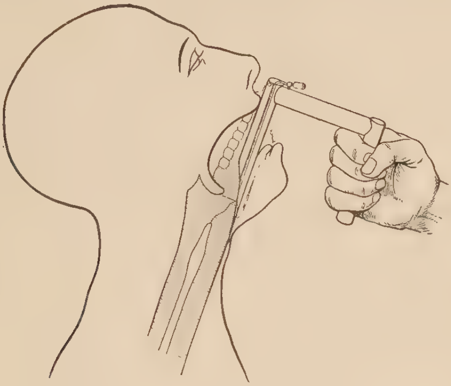

Fig. 55.—Schema showing correct position of patient and of the instrument in relation to the air-passages. The instrument should touch the upper teeth very gently, if at all. This is also the proper position for esophagoscopy (Chevalier Jackson).

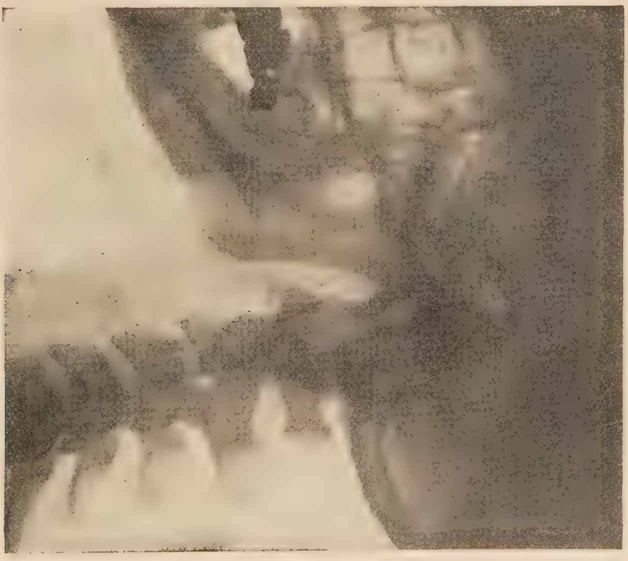

Fig. 56.—Correct position of the cervical spine for esophagoscopy and bronchoscopy. Radiograph by Dr. George C. Johnston (Chevalier Jackson).

In the dorsal recumbent position, the head should be squarely extended on the occipito-atlantal joint, not on the cervical spine. If the head is held in the position of Roser, the cervical vertebræ are curved, and the esophagoscope abuts almost vertically on the convexity and cannot be introduced with the introitus esophagi, according to Jackson (Fig. 57).

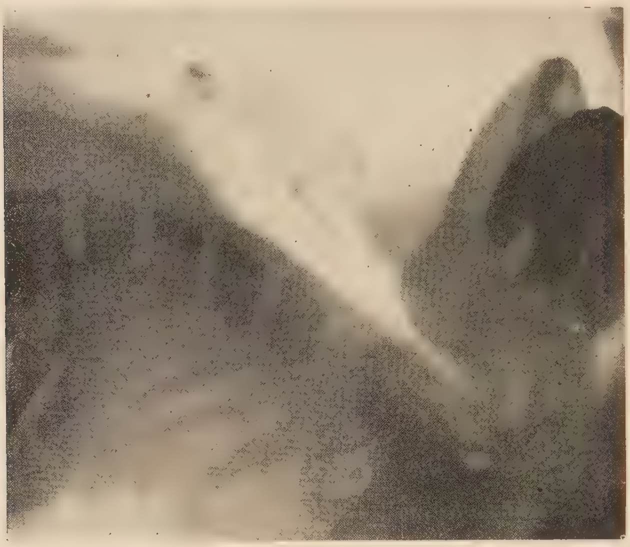

Fig. 57.—Curved position of the cervical spine in the Roser position, rendering esophagoscopy and bronchoscopy difficult or impossible. The devious course of the pharynx, larynx, and trachea are plainly visible. Radiograph by Dr. George C. Johnston (Chevalier Jackson).

The assistant is placed on the right side of the patient's head, on a stool of appropriate length, as though on a side saddle, his right leg beneath him in the kneeling position, his left foot supported on a stool 26 inches lower than the top of the table. His right forearm is passed beneath the patient's neck, supporting it; his right hand holds the

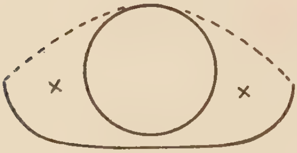

Fig. 58.

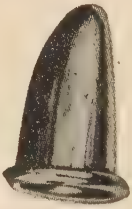

Fig. 59

Fig. 58.—Schema showing relation of the cricoid cartilage (the circle) to the posterior hypopharyngeal wall, in the dorsally recumbent patient, observer looking down the esophagus. The pyriform sinuses are at the postion marked ×.

Fig. 59.—Thimble gag or bite block for bronchoscopy and esophagoscopy (Jackson).

mouth-gag, drawing it strongly at, or in front of, the bregma, bending it backward and exerting a certain degree of upward pressure. The mouth-gag is inserted on the left side. The patient should have the tops of the shoulders clear of the table by from 4 to 6 inches.

If an attempt is made to pass the esophagoscope in the *middle line*, it *encounters the cricoid* cartilage. It should be inserted into the pyriform sinus (Fig. 58), preferably the right one, and thence into the esophagus. A bite block, preferably Boyce's (Fig. 59), should be used in preference to a mouth-gag—the so-called thimble gag. Jackson's armamentarium for esophagoscopy consists of the following instruments:

1 esophagoscope (10 mm. by 53 cm.) for adults.
1 esophagoscope (7 mm. by 45 cm.) for children.
1 adult's slide speculum.
1 child's slide speculum.
1 aspirator for the esophagoscope, to remove secretions.
1 specimen forceps, long and short (Fig. 60).
1 foreign body forceps.
3 Coolidge sponge-holders.

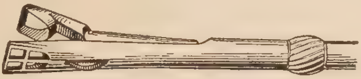

Fig. 60.—Specimen forceps tip to fit universal handle. The side jaw will bite into a flat lateral wall. The cross forms the bottom of a basket to hold the tissue removed.

In esophagoscopy for foreign bodies, the tube should be introduced without the mandrin, and every part be explored during its introduction. This avoids the danger of overriding the foreign body. The safest current for the light is a double dry battery, two sets of four cells each, in case one fails. Each set should have two binding posts and a rheostat. Ingalls holds that rheostats devised for adapting commercial circuits to tube work are dangerous when attached to a tube which makes a moist contact with tissues close to the course of the vagi. It is preferable, when possible, to determine the position of a foreign body in the esophagus by a radiograph before attempted removal through the esophagoscope. *Both lateral* and *anteroposterior radiographs* should be taken, as the foreign body may show in one and not in the other.

Plummer[1] recommends Brunning's modification of Killian's esophagoscope (Fig. 61), as it has a tip well adapted for easily passing the cricoid. Occasionally, a guide may be necessary, after the methods already described under Stenosis of the Esophagus.

Lerche[2] has devised an ingenious instrument for closing an open safety-pin impacted in the esophagus, and removing the same through the esophagoscope (Fig. 62).

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[1] Jour. Amer. Med. Assoc., Feb. 25, 1911.
[2] Ibid., March 4, 1911.

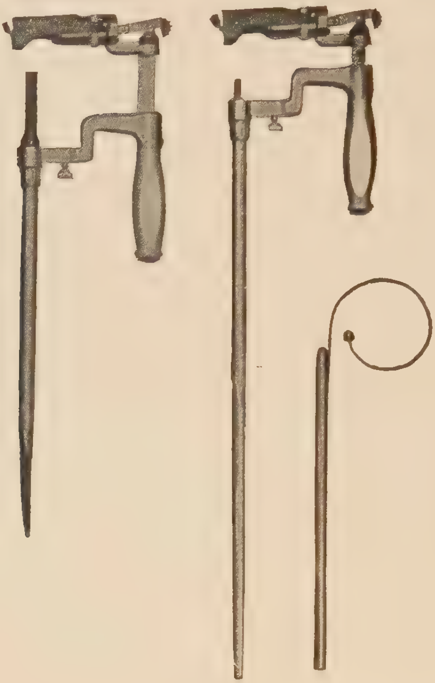

Fig. 61.—Brunning's modification of Killian's esophagoscope (Plummer).

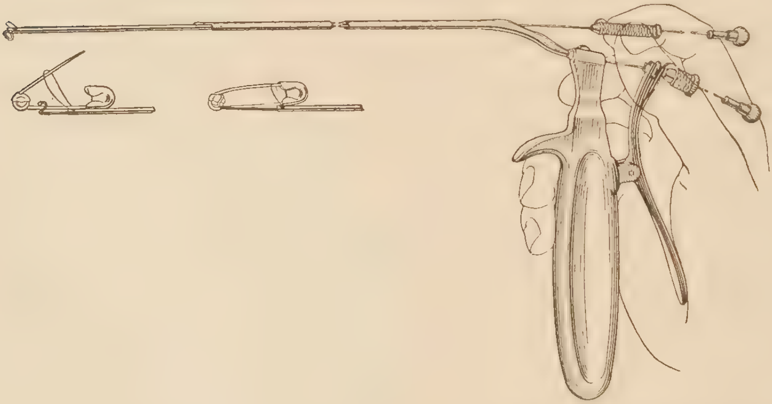

Fig. 62.—Instrument devised for closing open safety-pin and directing it through gastroscope (Lerche).

*Indications.*—Esophagoscopy is of chief value as an aid for the removal of foreign bodies. It may be employed for inspection of suspected ulceration and for topical application through the esophagoscope. In suspected cancer it should be used with caution.

*Contraindications.*—Aneurysm or recent hemorrhage.

**Gastroscopy.**—This method of examination of the gastric mucosa was inaugurated by Mikulicz in 1881.[1]

The general principle of the instrument is based on the cystoscope. Rosenheim has devised a new instrument, and Chevalier Jackson has carried on numerous investigations with gastroscopy. The latter has conclusively demonstrated that general anesthesia should be given for such an examination.

Chevalier Jackson's method with gastroscopy is described under his dorsal method of introduction of the esophagoscope. The instruments are identical, except that the gastroscope is longer, as in Fig. 63.

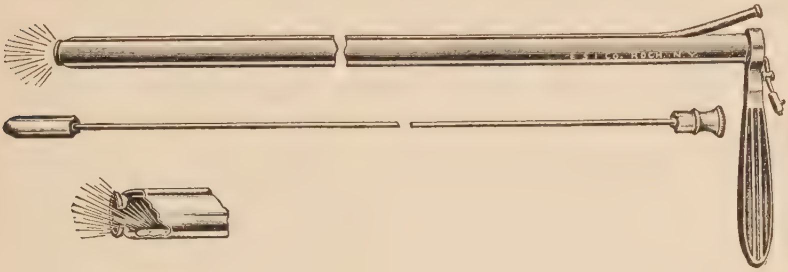

Fig. 63.—Chevalier Jackson's gastroscope.

Einhorn[2] has recently devised a gastroscope in which the lamp serves as an obturator. It can be pushed aside at the time of inspection.

I must confess that in cases of suspected cancer or ulcer I would not submit a patient to the certain degree of risk from the introduction of an instrument of the present type; and in milder cases of gastric disturbances I can see no advantage, except for the purpose of scientific study. It would seem that a large number of normal organs, as well as mild types of gastric disease, should first be investigated as a basis for comparison, and that the instrument should be improved upon before the method can be generally recommended.

The gastroscope is a valuable adjunct for the attempted removal of foreign bodies from the stomach, such as a tack, a pin, etc.

**Gastrodiaphany or Transillumination of the Stomach.**—Casenave, in 1845, first applied the method of transillumination to living tissues. In 1867 Milliot succeeded in transilluminating the stomachs of animals and experimented with the stomachs of cadavers, but to Max Einhorn, of New York, the credit is due of being the first to demonstrate transillumination of the stomach on the living subject and the practical value of gastrodiaphany. His instrument, which he denominates a gastrodiaphane, consists of a soft-rubber stomach-tube, at one end of which is fastened an Edison lamp. Conducting wires run through the tube to the battery, and there is a current interrupter at some distance from the tube. The lamp is inclosed in a glass bulb, to act as a reflector and prevent the action of heat on the stomach. He has the patient drink only 1 or 2 glasses of water, so as not to distend the stomach, inserts the light, and examines the case in a dark room, either in the sitting or in the recumbent position.

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[1] Wiener med. Presse, 1881, No. 45.

[2] Med. Record, June 11, 1910.

Heryng and Reichmann employ a modified tube, with a water-cooler about the lamp. Kuttner and Jacobson, under Ewald's direction, performed a great number of experiments.

These experimenters, together with Meltzing, are the chief foreign investigators with gastrodiaphany. Manges, Stockton, and many others have employed it. Among various gastrodiaphanes are those or Hemmeter, Lincoln, Solis-Cohen, Koplik, and Lockwood. To Lockwood we must credit a decided advance in the type of instrument—a fine, wire-bound cable (rubber insulated) and a small light, no larger than a 5-grain capsule. The cut of my instrument, the "circumscribing gastrodiaphane," will sufficiently explain the Lockwood instrument, after which it is modeled, with certain additions.

**The Circumscribing Gastrodiaphane.**—A series of observations with transillumination of the stomach suggested an improvement on the gastrodiaphanes in use. Manipulation of the tube after the electric light has entered the stomach frequently causes gagging and, at times, vomiting, interfering thus with the accuracy of the method. The cables of all the instruments were found unsatisfactory in cases of gastroptosis of great degree when we endeavored to explore carefully the pyloric end of the greatly dilated stomach. It was impossible to guide the light in a definite direction; it would sometimes pass to the right, sometimes to the left, and often it was necessary to draw it in and out a number of times for a distance of several inches.

The instrument I devised to overcome these drawbacks has a cable about 6 inches longer than the Lockwood gastrodiaphane, and is of about the same caliber (Fig. 64). The cable is more flexible for the space of $\frac{1}{4}$ inch at about the same distance from the light—in effect, a joint at this point. At the base of the light is attached an extremely thin accessory cable, covered with rubber. This runs parallel with the main cable and increases the diameter only a very slight degree. After introduction of the instrument the main cable is held firmly, and the accessory cable drawn upon. By turning the cable at the same time, the instrument can be guided in the desired direction. By manipulation of the accessory cable the main cable can be so bent that the light will explore the entire wall of the stomach anteriorly, and can be made to pass up to the pylorus and along the borders of the ribs. The lesser curvature is thus explored.

Care should be taken that the cables are parallel when passed into the stomach, and the accessory cable should be relaxed before withdrawal. The main cable, except at the joint near the light, is stiffer than the Lockwood light. Eight dry cells are employed with a rheostat. Wappler manufactures a small pocket battery with six cells which I have found excellent. An extra lamp should be carried. Water was the medium formerly employed.

A great advance in the technic of gastrodiaphany is the employment of fluorescent media.[1]

There are three such media found to be of value:

Bisulphate of quinin, 10 grains (0.6), in a pint of water. The addition of 5 minims (0.3) of dilute phosphoric acid intensifies its action. The same amount of dilute sulphuric acid may be substituted. The reaction of the quinin solution is acid and the fluorescence a very pale violet. Increased acidity intensifies its action and fluorescence disappears if the solution is rendered alkaline.

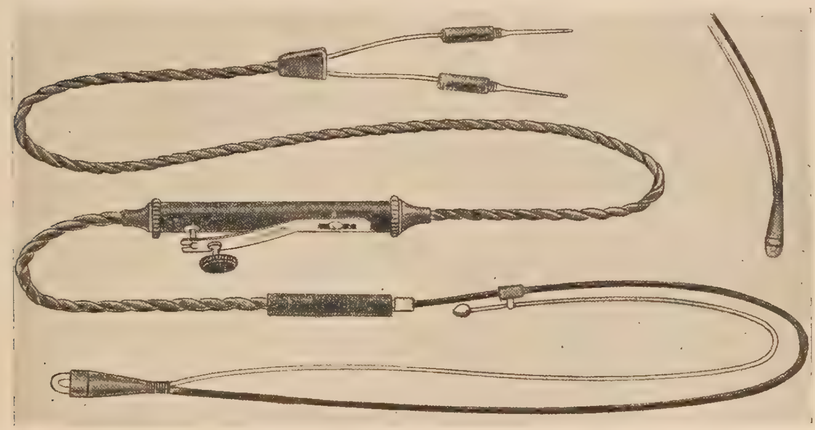

Fig. 64.—Kemp's gastrodiaphane (circumscribing).

Esculin. This is derived from the Æsculus hippocastanum (horse-chestnut), indigenous to Europe; 15-grain doses have been used in malaria. One can employ small doses, $\frac{1}{8}$ to $\frac{1}{2}$ grain (0.008–0.032), in a pint of alkaline solution, which gives a blue fluorescence. This preparation is difficult to secure and is expensive.

Fluorescein (phthalic anhydrid, 5 parts), a naphthalin product, and resorcin (7 parts), heated to 200° C. (392° F.). It is a reddish powder, faintly soluble in water, with a neutral action, and gives thus no fluorescence; soluble in alcohol and in alkaline media, it gives a green fluorescence like liquid opal. It has been employed to detect ulcers of the cornea. It can be secured from Merck & Co., and is extremely cheap.

The addition of glycerin intensifies the fluorescence, and the hydrochloric acid of the stomach must first be neutralized. The patient should first be given a glass of water (8 oz.—250 c.c.) in which 15 grains (1) of bicarbonate of soda have been dissolved. A second glass of water (8 oz.—250 c.c.) is then administered, in which are dissolved the same amount of sodium bicarbonate, 1 dram (4) of glycerin, and $\frac{1}{8}$ to $\frac{1}{4}$ grain (0.008–0.016) of fluorescein; 1 or 2 ounces of lime-water may be substituted for the sodium bicarbonate.

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[1] New York Med. Jour.; Philadelphia Med. Jour., Feb. 13, 1904; New York Med. Jour., August 6, 1904.

As we increase the fluorescein in strength, fluorescence diminishes and colorization begins. By means of fluorescent media I have found it possible to illuminate the stomachs of fat or muscular subjects that were formerly unsatisfactory, and to examine for tumors and for the location of the stomach with greater accuracy. The brilliancy of the illumination is markedly increased.

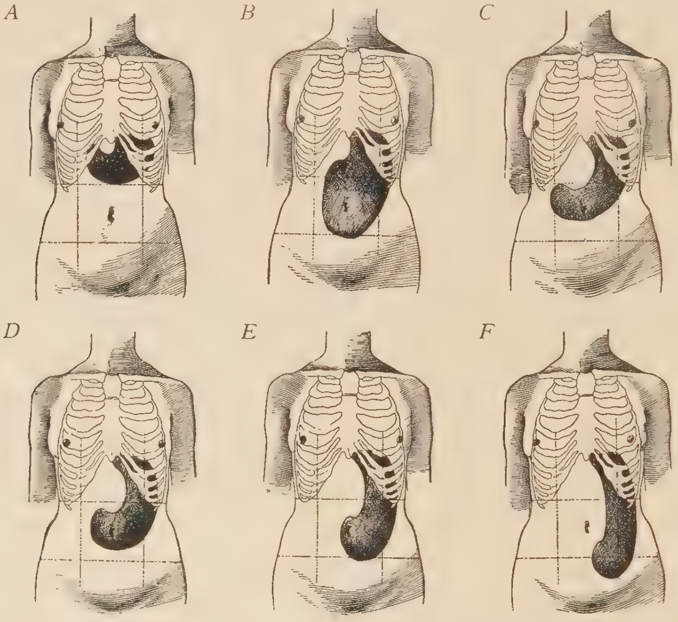

Fig. 65.—Gastrodiaphany: *A*, Normal stomach; *B*, dilated stomach; *C*, *D*, *E*, *F*, varying degrees of gastroptosis (Rose and Kemp).

Examination of the urine of patients who have taken fluorescein shows no deleterious effects—no albumin, no sugar, no casts. The fluorescein acts in an alkaline medium, and free acid destroys fluorescence, yet on catheterization of these patients greenish fluorescent urine is obtained one hour after the administration of fluorescein solution, and this condition persists for about four hours. The acidity of the urine is not due to the presence of free acid. With fluorescein solution we have an additional means of testing the permeability of the kidneys.

The technic of gastrodiaphany is as follows: The patient's stomach should be empty. He is given a glass (8 oz.—250 c.c.) of the alkaline solution, and then a second glass (8 oz.—250 c.c.) of the fluorescein solution. I frequently give *an extra half or even two additional glasses of water*, especially in the suspected cases of dilatation or ptosis or in stout subjects. In the latter, gastrodiaphany is not as satisfactory, but by pressing on the abdominal wall, the outlines can be secured.

A dark room gives the most satisfactory results. It can be devised by pinning blankets across the windows. The patient can also be examined in a light room by covering him from neck to feet with a dark blanket or black gown, and the examiner looking through an opening therein.

The gastrodiaphane is introduced by gaslight or candlelight, the patient sitting opposite in a chair, with the abdomen exposed. The electric current is turned on and the room darkened. The patient should then stand up, as this position is preferable. It is my custom to mark out the anatomic regions on the abdomen of each case with blue pencil, and then draw the outlines of the stomach during transillumination.

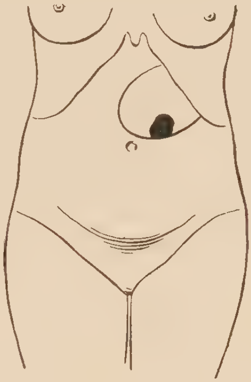

Fig. 66.—Cancer of greater curvature. Gastrodiaphany.

With gastroptosis, the lesser curvature can be determined. In some cases the stomach will be bottle-neck above, with the base below; or somewhat pear shaped, the narrow part showing above, as the light disappears beneath the ribs. With a dilated stomach the transverse diameter of transillumination is nearly the same throughout; as we withdraw the light it begins to narrow just below the tip of the ensiform. If we illuminate in the dorsal position, the light hardly shows at all; it becomes clearer as the patient gradually sits erect, and is most marked in the standing position. This substantiates the view of Meltzing, who states that in the dorsal position only a portion of the stomach is in contact with the abdominal wall, and it demonstrates the necessity of the standing position for accurate illumination.

In Fig. 65 are shown a normal stomach, the dilated organ, and several degrees of gastroptosis. There is no question but that ptosis of the stomach exists from a very slight to an extreme degree. Tumors or thickenings of the anterior wall of the stomach, or of the lesser or greater curvature, or anterior surface of the pylorus, appear as a dark area projecting into or surrounded by a light zone (Fig. 66).

By the circumscribing gastrodiaphane the entire contour of the stomach is determined. The older instruments show only the lower segment clearly.

## RONTGEN RAYS (*X*-RAYS) IN THE DIAGNOSIS OF DISEASE THE ESOPHAGUS AND STOMACH

Though unquestionably the discovery of the *x*-rays constitu one of the greatest advances in medicine in recent times, space wi only allow the writer to refer briefly to those conditions in which he believes this method to be indicated as an aid to diagnosis. In discussing the subject one must distinguish between the use of the *x*-rays for the purpose of scientific study and their practical value as an aid to diagnosis. They seem to the author to be recommended in some cases in which they are unnecessary; in addition, being an expense to the patient, with a certain element of risk.

**The Esophagus.**—By means of the *x*-rays the scientific study of the motor functions of the esophagus has been rendered possible. One can readily make a diagnosis of stenosis of the esophagus or of a diverticulum by the methods already described on page 50. The *x*-rays after the ingestion of bismuth are of value as an adjunct. One can thus readily determine the contour and relations of a diverticulum to the esophagus (Figs. 22, 23, and 24, p. 52), a fusiform dilatation, a stricture, abnormalities due to adhesions, fistulæ, alternations of outline, and pressure of aneurysm or tumor. The author advocates the use of *x*-rays when possible, though generally one can make the diagnosis without their use. The position of a foreign body can usually be determined by direct inspection through the esophagoscope, and can be at the same time removed. A preliminary röntgenograph is preferable when possible.

**The Stomach.**—From a scientific standpoint, the study of the stomach with the *x*-rays has proved of great value. It has thus been demonstrated that the fundus is vertical and the pyloric portion is transverse, and that the viscus does not lie transversely across the abdomen, as was formerly supposed. Cannon has also made an interesting study of the motility of the stomach by means of the *x*-rays, and also of the relative rapidity of the exit of the carbohydrates, proteins, and fats from this organ.

Rieder, Pfahler,[1] Cole, and Caldwell demonstrated by the cinematographic method normal gastric peristalsis, and the part these waves play in the diagnosis of carcinoma of the stomach. They conclude that the so-called special sphincter of Holzknecht, of the pyloric antrum, represent merely the point of maximum depth of the waves and that there is no true antrum. From a practical standpoint Röntgenography is of great value for the detection of a foreign body lodged within the stomach and for demonstrating its location. The possibility of its removal through the gastroscope, or the location of an incision for its removal, often depends upon the information secured by this means.

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[1] Pfahler, New York Med. Jour., Aug. 21, 1909; Med. Rec., March 25, 1911; New York Med. Jour., May 6, 1911; Jour. Amer. Med. Assoc., June 17, 1911.

Hour-glass stomach can be determined by means of the $x$-rays (Fig. 67). The diagnosis can be made by the methods described under that subject, but it is advisable to confirm the same by Röntgenography when possible.

Röntgenography has been recommended for the purpose of locating the position of the stomach. The method is expensive for the patient, and *no information* can be secured that cannot be obtained by the usual methods of physical examination.

The Röntgen photograph shows the greater curvature and part of the body of the stomach when taken in the standing position, in which posture the greatest prolapse of the lower border occurs. By comparing this with fluorescent gastrodiaphany with the circumscribing gastrodiaphane, one finds the lesser curvature is delineated at a higher level in many cases by the latter method. The bismuth solution gravitates to the lower part of the stomach and the lesser curvature is not always as low as delineated by the $x$-rays. The location of the lesser curvature is the important feature in the differential diagnosis between gastroptosis and dilatation of the organ. The writer has already stated that mobility of the kidney plus descent of the lower border of the stomach is diagnostic of gastroptosis, while descent of the lower border alone shows simply dilatation of the organ.

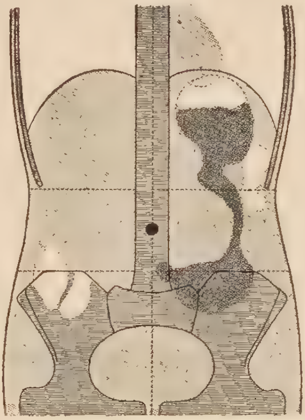
Fig. 67.—Hour-glass stomach.

**The Diagnosis of Carcinoma.**—Holzknecht, Jonas, Pfahler, and Cole particularly advocate the value of the $x$-rays for the early diagnosis of carcinoma of the stomach. Pfahler,[1] however, makes the following interesting statement: "Based upon a rather large experience in the Röntgen diagnosis of carcinoma of the stomach, it is my opinion that we have in this method the most positive means yet devised for the recognition of carcinoma in any stage of the disease. It stands *next to an exploratory operation*." This *last is significant*. The value of the method of diagnosis depends on the fact that carcinoma of the stomach modifies the outline, position, or lumen of the organ, or interferes with the peristaltic waves, the motility of any part, or obstructs the passage of food. The earliest evidence will be some interference with the peristaltic waves. In Fig. 68 is a Röntgenograph of carcinoma.

Fluoroscopy is the most valuable feature of the method, but the Röntgenograph is also necessary. There is considerable danger in fluoroscopy to the operator and also to the patient, unless *in the hands of an expert.* The writer would *recommend the physician* to avail himself of the method if he can secure the services of such. There are many who can take an excellent Röntgenograph, but comparatively few at the present writing *qualified by experience* to correctly interpret the fluoroscopic appearances diagnostic of early cancer. As a rule, therefore, for the majority of practitioners, an *early exploratory* incision is preferable. Pfahler describes the *x*-ray method in an interesting article in the Medical Record, March 25, 1911.

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[1] Med. Rec., March 25, 1911; N. Y. Med. Jour., May 6, 1911; Jour. Amer. Med. Assoc., June 17, 1911.

Barclay and others have demonstrated that spasmodic strictures occur with gastric ulcers, and that the latter can often be more readily recognized by fluoroscopic demonstration of this condition than by the retention of bismuth in the ulcer.

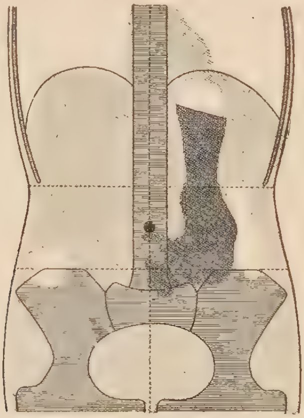

Fig. 68.—Carcinoma of pylorus. Stomach is dilated, mostly vertical, reaching to false pelvis. Some evidence of peristalsis in walls of vertical part. The horizontal part extends to the middle line, and ends with a peculiar serrated border (characteristic for carcinoma).

The author believes, however, that the ordinary methods of diagnosis of gastric ulcer, particularly the presence of occult blood and pus in the gastric contents, are usually sufficient.

**Perigastric Adhesions.**—Cole has demonstrated the effects of adhesions on the peristaltic waves of the stomach cinematographically. The lesser curvature is usually involved. Pfahler shows that the normal position of the stomach and duodenum is modified by adhesions to the gall-bladder—in other words, that these organs are pulled upward and to the right. With chronic cholelithiasis and cholecystitis, the swelling of the liver and gall-bladder brings them nearer the stomach and duodenum, and the pericystitis leads to adhesions. When the swelling is reduced, the adhesions pull the stomach upward and to the right (Fig. 69). In such cases the clinical symptoms appear during digestion, when the motor functions of the organs are at their height, especially when the emptying of the gall-bladder has been interfered with. The $x$-rays, therefore, may be of value as an aid to diagnosis. Bismuth subnitrate has been generally abandoned on account of the possibility of a toxic effect, methemoglobinemia having been reported as a result. Bismuth subcarbonate, 1 ounce in a glass of kefir, fermillac, or koumiss, is excellent for $x$-ray work.

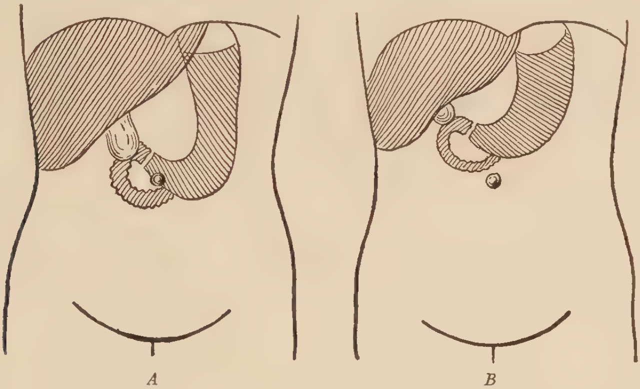

Fig. 69.—A.—Stomach in normal position, but the liver and gall-bladder swollen and thereby approaching more closely the stomach and duodenum, and more easily permitting the formation of adhesions to these organs.

B.—Liver and gall-bladder returned to their normal position, the stomach and duodenum drawn upward and to the right by adhesions (Pfahler).

Bismuth oxychlorid may be substituted. Cole and Einhorn[1] have advocated radiograms of the digestive tract by inflation with air, but the author does not believe the method as satisfactory as by the older technic.

The determination of gastric atony by a study of the peristaltic waves (*i. e.*, their absence) is interesting scientifically, but simple methods only are necessary for the purpose of diagnosis.

## RADIUM TRANSILLUMINATION OF THE STOMACH

This method was first suggested by Einhorn[2] with his radiodiaphane (Fig. 70), a rubber-covered glass capsule containing 0.05 gm. bromid of radium (Curie, 1,000,000 strength), an inflating bulb, and Kahlbaum's fluoroscope. The transillumination is very

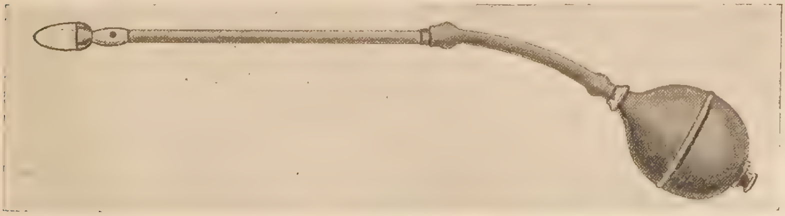

Fig. 70.—Einhorn's radiodiaphane.

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[1] New York Med. Jour., Oct. 8, 1910.

[2] Med. Rec., July 30, 1904.

faint, and the results not as clear as from gastrodiaphany. The expense is considerable. At the present stage of its development I would not recommend it as of practical value.

## RADIUM PHOTOGRAPHS OF THE STOMACH

The same may be said of this procedure devised by Einhorn.[1]

The length of exposure to radium is never less than an hour, and, preferably, longer to secure results.

This is decidedly objectionable, as severe burns are possible from radium. If photographs are to be taken, the *x*-rays are safer and give more definite results.

## CONCLUSIONS

The following methods I have found from experience to be most practical for general use:

**Inspection.**—By this method the peculiar shape of the abdomen, suggestive of gastroptosis, can at once be determined.

**Palpation.**—If movable kidney be present, it is pathognomonic of gastroptosis, especially if the lower border of the stomach lies abnormally low. Sensitive areas can also be determined, and often the presence of a tumor.

**Percussion.**—The scratch method of auscultatory percussion is serviceable in mapping out the stomach, as is also auscultatory percussion.

**Splashing Sound.**—If not present, it can be artificially produced, and is most *valuable in determining the lower border of the stomach.*

**Dehio's Method.**—Additional water can be given, if desired, and by percussion the observations determined by the splashing sound can be substantiated for accuracy.

**Gastrodiaphany.**—This can be employed to differentiate in doubtful cases between ptosis and dilatation, and is of value in accurately mapping out the stomach before surgical operation. By it one can at times determine the presence of a tumor at an early stage.

**Inflation.**—This method, especially by distention with carbonic acid gas, is an aid in mapping out the stomach and in determining the position of the upper as well as of the lower border.

***x*-Rays.**—Are confirmatory of stenosis or diverticulum of the esophagus or of the presence of a foreign body. They will also determine the latter in the stomach and the presence of hour-glass stomach. In the hands of an expert, they assist in the determination of perigastric adhesions, carcinoma, and ulcer.

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[1] Archives of Physiological Therapy, Sept., 1905.

## CHAPTER VI

# EXAMINATION OF THE FUNCTIONS OF THE STOMACH

The determination of the functions of the stomach—secretory, motor, and absorptive—is of importance for accurate diagnosis. Of the examination of the secretory function I will first speak.

Ewald and Boas have demonstrated that gastric secretion begins as soon as food enters the stomach, and continues until the chyme has passed into the intestines. The secretion is diminished toward the last. Examination at various periods after taking food will give different results, and it is necessary to examine the gastric contents at a definite time during the height of digestion. It is desirable that a definite test-meal should be administered.

### TEST-MEALS

**Riegel's Test-dinner.**—Riegel's test-dinner is the oldest advocated. This consists of a plate of meat broth (about 400 c.c.); a beafsteak weighing from 150 to 200 gm. (5–7 ounces); 50 gm. (1½ ounces) of mashed potatoes; and a roll (35 gm.).

The average time one should aspirate the stomach-contents is about four hours after this meal.

**Ewald's Test-meal.**—This consists of about 6 ounces (175 gm.) of finely chopped meat; stale bread, 35 gm., and butter, to be taken three hours before withdrawal of the stomach-contents.

This is practical as regards quantity, as by some the test-meal of Riegel is considered large in amount.

**Test-meal of Germain Sée.**—The patient is given 3 to 5 ounces (100–150 gm.) of white bread; 2 to 3 ounces (60–80 gm.) of finely chopped meat, and a large glass (300 c.c.) of water, and the contents examined two hours later.

**Klemperer's Test-meal.**—This consists of 1 pint (500 c.c.) of milk and 2 rolls (70 gm.), given on an empty stomach. Examination two hours later.

**Test-breakfast of Ewald and Boas.**—This is given in the morning in the fasting condition, and consists of 1 to 2 rolls (35–70 gm.) and 1 cup (300–400 c.c.) of tea or water. Examination one hour later.

**Dry Test-meal.**—Boas has suggested a dry test-breakfast, consisting of a roll without water, as giving a more accurate index of gastric secretion. In cases of disordered motility the dry method is probably more accurate, but from a practical standpoint there is no advantage in the method. Chace[1] reports a recent series. Some prefer it in differentiating between hyperacidity and hypersecretion.

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[1] Jour. Amer. Med. Assoc., July 1, 1911.

**Boas' Test-breakfast.**—One ounce of rolled oats boiled in 1 pint of water, with salt to taste.

Boas advocates this, as it *contains no lactic acid*, and believes it should be employed when an *accurate test for this acid* is desired.

Two shredded wheat biscuits with water (300 c.c.) or a pint bowl of granose have been recommended as a convenient substitute.

Sahli, Jaworski, and others have suggested modifications, but they possess no advantages.

The test-meals that are in chief use are the Riegel test-dinner and Ewald-Boas test-breakfast. The latter is easily procured and can be administered during office hours—*which is the most accurate method.* It is easy to recognize therein remnants of food from the previous day. The test-dinner gives better results as regards the investigation of the microscopic appearances and the study of the motor functions. *The test-breakfast in many cases will be sufficient.*

It is often of service to administer a special test-dinner at 7 P. M. and the test-breakfast in the office at 7.30 to 8.00 A. M. It will be described under Testing the Motor Functions.

I agree with Fleiner that tea is to some an irritant. In addition, it is not of stable strength, and water is preferable. The average slice of bread from the loaf weighs 30 gm. I employ *usually 2 slices (60 gm.) of bread* and *300 to 350 c.c. of water*, the latter by preference. One must allow for a patient of small physique and poor nutrition, for in such cases he cannot or will not take this quantity.

The *diagnosis* in most cases *should not be made from a single examination* of the gastric contents.

E. L. Eggleston[1] reports some interesting variations of the gastric juice after similar test-breakfasts in the same patient. One should make several tests and take a general average. In some patients it would be probably preferable to employ the test-dinner as well as test-breakfast. Moreover, if the patient has been under medical treatment by another physician, all internal medication should be stopped for several days previous to examination. With a very nervous patient, it is preferable not to tell her that the stomach-tube is to be passed until about to do so, otherwise the nervous influence would markedly affect the results. The gastric secretion (appetite juice) is undoubtedly greater after a dinner of a character agreeable to the patient than following an unattractive meal, consisting of bread and water. All these factors must be considered.

## METHOD OF ASPIRATION OF THE GASTRIC CONTENTS

The selection of the proper type of tube is important. It should be of soft rubber, of a caliber of 30 to 32 French, to allow free exit of contents; have an opening at the tip, and one or even two lateral openings, as in Fig. 71, or the Ewald tube (Fig. 72).

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[1] Observations on the Variability of the Gastric Juice, New York Med. Jour., Oct. 29, 1910.

**Position of Patient and Operator.**—The patient sits upright on a chair and is protected by a sheet or towel. False teeth should be removed. The operator stands in the position as depicted in Fig. 73, and passes the tube along the roof of the patient's mouth. The advantages of this method are described under Lavage.

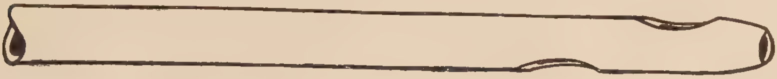


Fig. 71.—Aspirating tube.

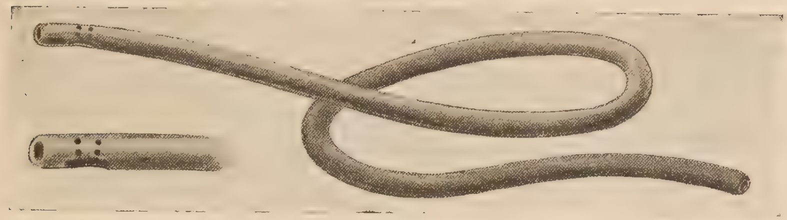


Fig. 72.—Ewald's tube.

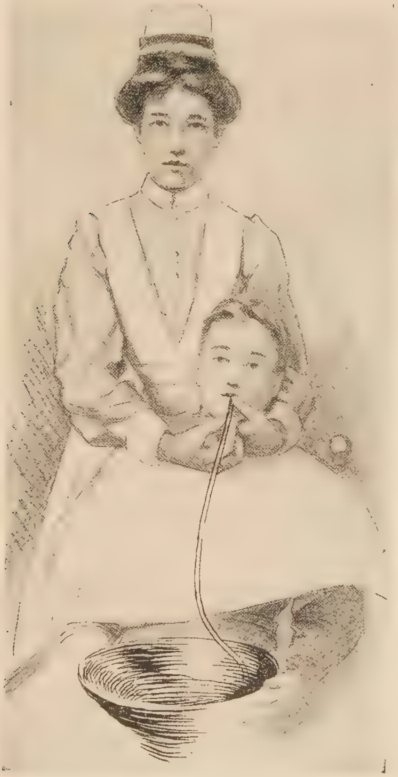


Fig. 73.—Correct position for passage of aspirating tube.

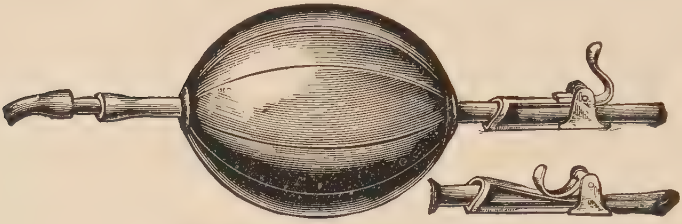


Fig. 74.—Boas' aspirating bulb.

Fig. 75.—Aspirating bulb.

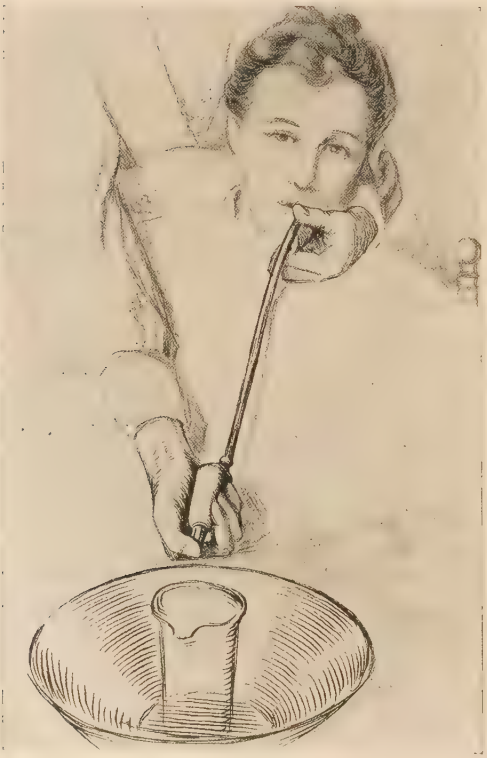


Fig. 76.—Filling the bulb.

The tube should be moistened in warm water and introduced about 20 inches until resistance is felt, when it should be slightly withdrawn. In the event of gastroptosis or dilatation the distance would be greater. A Politzer bulb can be employed for aspiration.

*Boas' Method.*—The Boas aspirator (Fig. 74), which consists of a rubber bulb having two soft-rubber ends and provided with a clamp, is attached to the stomach-tube by a piece of glass tubing. The clamp is fixed, the bulb compressed and then released, and thus filled up; the clamp is opened, and the contents, by compression of the bulb, forced into a bottle, glass, or some other receptacle. This is a simple method of aspiration. Other devices with a vacuum bottle, mouth suction, forcing air through a double tube, etc., have been suggested, but they are more complicated and unnecessary.

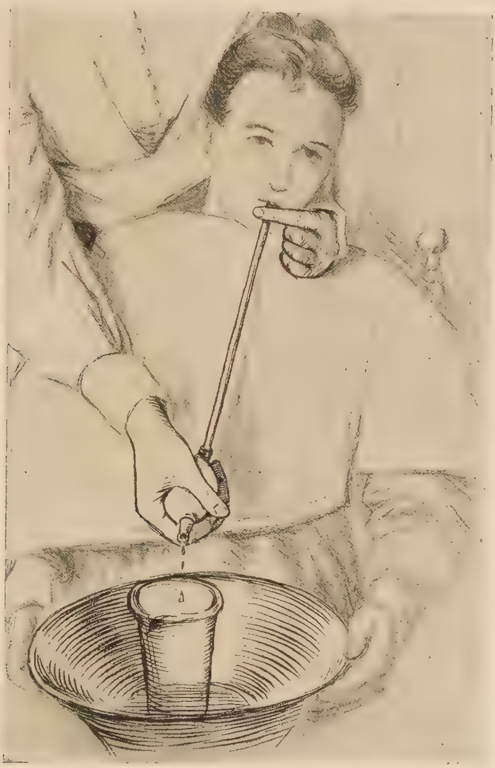


Fig. 77.—Second step of aspiration.

**Ewald-Boas Expression Method.**—This consists in having the patient exert pressure upon the stomach by means of his abdominal muscles. He should first inspire deeply, and then compress the abdominal walls in the same manner as during defecation. The pressure expels the gastric contents through the tube into a receptacle. This method is considerably employed, and when successful, is unquestionably excellent. In cases of marked atony, both of the stomach and abdominal walls, in excessive dilatation, and at times from plugging of the stomach-tube, the expression method is a failure.

**Author's Method.**—It is my custom to attach to the stomach-tube a bulb without valves, such as is depicted in Fig. 75. The patient is then directed to express the contents. If the tube becomes stopped, the thumb is placed over the open end, the bulb squeezed, and the obstruction immediately relieved. This obviates blowing through or removing the tube.

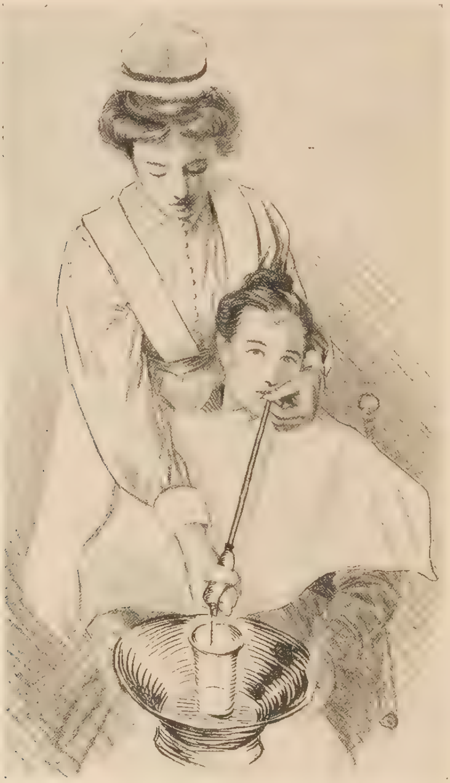


Fig. 78.—Final step of aspiration.

If expression fails, them immediate aspiration is resorted to. The tube is pinched near the teeth, the bulb squeezed, and the thumb placed over the open end. The stomach-tube is then released, and the vacuum in the tube allowed to fill with gastric contents, as in Fig. 76.

When the bulb is filled, the stomach-tube is again pinched, the thumb removed from the bulb, and the contents gradually expressed into the receiving vessel, as in Fig. 77.

The final step is depicted in Fig. 78. This process is repeated until the gastric contents are removed.

## EXAMINATION OF THE INGESTA

Before chemic examination of the ingesta is begun, the quantity aspirated should be carefully measured. After the Ewald-Boas test-breakfast one may expect to secure on an average 50 to 75 c.c. of contents, and if 100 c.c. or more be present, this would be suggestive of motor insufficiency. A large quantity of gastric contents (350 to 400 c.c.) four hours after the test-dinner would also be a suspicious circumstance, and further tests should be made, as described under Examination of Motor Functions. The quantity of the residue, therefore, has a chief bearing on the motor function, though in hypersecretion abnormal quantities are found.

Macroscopic inspection gives considerable information. After the test-breakfast, in some cases large undigested pieces of bread are brought up; in others, remnants of bread that are nearly digested or only slightly digested; and in others, a fine fluid mushy mass. These findings are at once suggestive. With Riegel's test-meal the differences are more pronounced; the mass may be fine, uniform, and mushy, containing no coarse elements; or there may be coarse undigested meat-fibers.

Mucus, blood, and bile are readily visible. In some cases the gastric contents, when placed in a glass vessel, forms three layers: at the bottom, fine starchy material; next, cloudy fluid; and on top a foamy layer, which latter is evidence of gaseous fermentation.

**Chemic Examination.**—The aspirated gastric contents should be filtered, preferably through filter-paper. If this is not at hand, several layers of cheese-cloth or gauze can be employed.

From a practical point of view, the most important feature to determine is the content of hydrochloric acid during the height of digestion. If free hydrochloric acid be present, it is then necessary to find out whether the secretion is normal, increased, or deficient. If it is deficient in amount or absent, the digestive power of the stomach is deficient.

When free hydrochloric acid is present, the determination of pepsin is unnecessary; in fact, it is often present, even if free hydrochloric acid is absent. In such event, if the gastric contents are acidified with sufficient hydrochloric acid and digestion of albumin then occurs, this is evidence of sufficient pepsin formation. In cases complicated by the absence of free hydrochloric acid the examination for pepsin should be carried out.

For a complete chemic analysis the following tests should be performed:

1. Reaction.

2. Total acidity.
3. Free hydrochloric acid.
4. Combined hydrochloric acid.
5. Total hydrochloric acid.
6. Lactic acid.
7. Propepton.
8. Pepton.
9. Pepsin.
10. Rennet.
11. Dextrin.
12. Erythrodextrin.
13. Achroödextrin.

Before describing the tests it is well to remember the findings of the normal gastric juice after the test-breakfast for a basis of comparison.

Normal gastric juice is of acid reaction; total acidity, 40 to 60 (0.15–0.21); free hydrochloric acid, 25 to 50 (0.1–0.2 per cent. approximately); propepton, small amount; pepton more; pepsin and rennet present; sugar and achroödextrin present; erythrodextrin present in small amount or absent; dextrin absent.

Some patients may have free hydrochloric acid within the above normal limits, and yet suffer from the symptoms of hyperchlorhydria, while others may have free hydrochloric acid as high as 100+ and present no symptoms at all. Individual peculiarities must be considered.

**Reaction.**—This is determined by means of blue and red litmus-paper. If the filtrate is acid, it turns the blue paper red; and if alkaline, the red turns to blue; neutral gastric contents cause no change.

**Test for Free Hydrochloric Acid.**—Numerous coloring-matters when exposed to the action of weak solutions of hydrochloric acid undergo changes, and have been employed as tests for its presence. Those that are of greatest practical value I will describe shortly.

There has been considerable dispute as to the respective superiority of these tests. Though organic acids, when present in considerable quantities, may give these color changes, yet they are not as sensitive to organic acids as to mineral acids. I agree with Riegel that this danger is practically negligible. As a precaution it is well to employ one of the following qualitative check tests, which react only to free mineral acids and not to the organic acids.

In addition, the test for lactic acid should be performed.

**The Phloroglucin-vanillin Test** (**Günzburg's**).—This reagent consists of 2 grams of phloroglucin and 1 gram of vanillin dissolved in 30 grams of absolute alcohol. An equal number of drops, 1 or 2 each, of this and the gastric juice are placed on a porcelain dish and mixed with a glass rod. The dish is then held over an alcohol lamp and the fluid allowed to evaporate slowly. A cherry-red color appears, as in Fig. 79, if free hydrochloric acid be present. If there

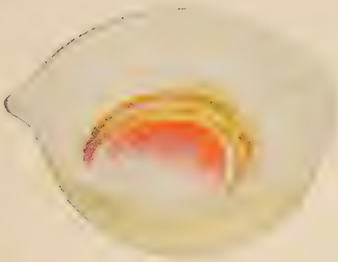


Fig. 80.—Resorcin test. Color reaction.

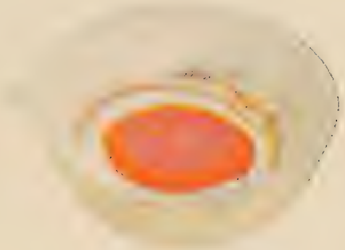
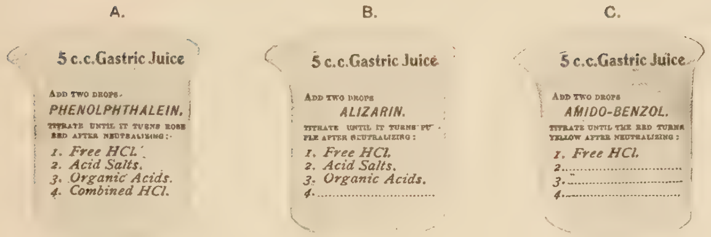


Fig. 79.— Phloroglucin-vanillin test. Color reaction.



A.

5 c.c. Gastric Juice

ADD TWO DROPS·
PHENOLPHTHALEIN.
TITRATE UNTIL IT TURNS ROSE RED AFTER NEUTRALIZING:·

1. Free HCl.
2. Acid Salts.
3. Organic Acids.
4. Combined HCl.

B.

5 c.c. Gastric Juice

ADD TWO DROPS
ALIZARIN.
TITRATE UNTIL IT TURNS PURPLE AFTER NEUTRALIZING:

1. Free HCl.
2. Acid Salts.
3. Organic Acids.
4. ..............................

C.

5 c.c. Gastric Juice

ADD TWO DROPS
AMIDO-BENZOL.
TITRATE UNTIL THE RED TURNS YELLOW AFTER NEUTRALIZING:

1. Free HCl.
2. ..............................
3. ..............................
4. ..............................

Fig. 81.

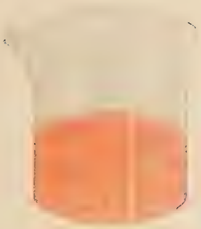


Fig. 82.—Phenolphthalein end-reaction. Total acidity.

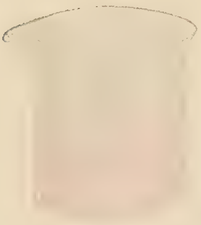


Fig. 84—Sodium alizarin sulphonate end-reaction.

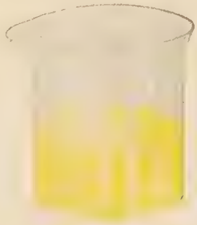


Fig. 83.—Dimethyl-amido-azobenzol end-reaction.

are only traces, then there is a rose tint at the margin. If hydrochloric acid is absent, the color varies from yellow to brown.

This test responds to *free hydrochloric acid and not to organic acids.* The solution is *unstable,* and should be preserved in *a dark glass bottle.* It is advisable to make a fresh solution frequently.

**The Resorcin-sugar Test (Boas).**—Five grams of resorcin and 3 grams of cane-sugar are dissolved in 100 c.c. of alcohol. Equal drops of this reagent and gastric juice are slowly evaporated to dryness, without burning, in a porcelain dish or a butter-dish. If free hydrochloric acid be present, a rose-red color appears, which fades on cooling (Fig. 80). It responds to hydrochloric acid only. It is nearly as delicate, more easily obtained, less expensive, and more stable than Günzburg's test.

After performing one of these check tests it is preferable in all cases to test quantitatively for acidity; and for this purpose I prefer *Töpfer's method.*

The qualitative method gives no basis for scientific accuracy.

**Töpfer's Method.**—This method determines quantitatively: Total acidity; free hydrochloric acid; combined hydrochloric acid; total hydrochloric acid, and acid salts quite accurately for clinical purposes. In routine examinations it is rarely necessary to determine more than total acidity and free and combined hydrochloric acid. If free acid is absent, then the pepsin and rennet tests should be made.

*Solutions Required.*—(1) One per cent. alcoholic solution of phenolphthalein (colorless).

(2) One per cent. aqueous solution of sodium alizarin sulphonate (opaque brownish yellow).

(3) Five-tenths per cent. alcoholic solution of dimethyl-amido-azobenzol (yellowish red).

(4) Decinormal solution of NaOH to titrate the gastric juice; 1 c.c. of tenth-normal NaOH neutralizes 0.00365 hydrochloric acid.

Töpfer's method depends upon the different sensitiveness of three-color end-reagents to the various constituents of the gastric juice.

*Method.*—Though 10 c.c. of the filtrate are usually employed, I have illustrated the method on the basis of 5 c.c., as it is often practically found impossible to carry out all the tests when using larger quantities. The methods are equally correct.

In each of the three beakers (*A*, *B*, and *C*, Fig. 81) are placed 5 c.c. of the filtered gastric contents.

To *beaker A* are added 1 or 2 *drops of the phenolphthalein solution,* which is used as an indicator of the *total acidity.*

This body turns red-pink or red as soon as the fluid becomes slightly alkaline, after the addition of the sodium hydroxid.

To *beaker C* we add 1 to 2 *drops of the dimethyl-amido-azobenzol solution.*

A reddish-pink or cherry-red color develops if free hydrochloric acid be present, depending on the degree of acidity. After titration with sodium hydroxid, the end-reaction is a pale orange yellow.

To *beaker B* is added 1 or 2 *drops of the sodium-alizarin-sulphonate solution.* After sufficient sodium hydroxid is added, a violet color (the end-reaction) appears.

The titration with sodium hydroxid is performed from a graduated pipet or, preferably, a buret, supported on a stand. The latter should be graduated to $\frac{1}{5}$ or, by preference, to $\frac{1}{10}$ c.c.

The elements combining the total acidity in beaker *A*, Fig. 81, are free hydrochloric acid, acid salts, combined hydrochloric acid and organic acids, which respond to the phenolphthalein test.

In beaker *B* are free hydrochloric acid, acid salts, and organic acids, responding to the alizarin test.

Hence, as Töpfer claims, to find the *combined hydrochloric acid*, one must subtract the *acidity of B* from the total acidity of *A*.

In beaker *C* we have free hydrochloric acid alone, which responds to the dimethyl-amido-azobenzol test.

In the absence of organic acids, the acidity of *C* subtracted from the acidity of *B* will give the acid salts.

*Total Acidity.*—The beakers being thus prepared, titration of beaker *A* (total acidity) is begun with the sodium hydroxid solution. As this is added, the reddish color appears, but it disappears as the fluid is agitated. The procedure should be continued until a permanent red-pink or reddish color is present, as in end-reaction (Fig. 82).

As the degree of acidity is expressed by the number of cubic centimeters of a decinormal solution of sodium hydroxid required to saturate or make slightly alkaline 100 c.c. of the gastric contents, and as only 5 c.c. of the latter was employed (which is $\frac{1}{20}$ of the total quantity), the number of centimeters of the sodium hydroxid necessary to produce the end-reaction must be multiplied by 20.

Thus if 3 c.c. sodium hydroxid produced the end-reaction in 5 c.c. of the filtrate, the total acidity would be 3 × 20, or 60+.

Multiply this figure of acidity by 0.00365, and we have the percentage of total hydrochloric acid, or $60 \times 0.00365$ equals 0.219 per cent.

*Free Hydrochloric Acid.*—Commence titration of beaker *C*, to which the dimethyl-amido-azobenzol solution has been added, and continue titration until the solution becomes a pale lemon yellow, as in Fig. 83.

As saturation with the decinormal sodium hydroxid solution was computed on the basis of 100 c.c. of gastric contents, and only 5 c.c. or $\frac{1}{20}$ were tested, the number of cubic centimeters of sodium hydroxid solution required to produce the end-reaction must be multiplied by 20. Thus, if 2 c.c. of this alkaline solution will produce this result, we must multiply it by 20, and we say the free hydrochloric acid is 40+.

To compute free hydrochloric acid in percentage, multiply $40 \times 0.00365 = 0.146$ per cent.

*Combined Hydrochloric Acid.*—Commence titration of beaker *B*, to which the sodium-alizarin-sulphonate has been added, and continue the process until the end-reaction, the violet color, as in Fig. 84, occurs.

As only 5 c.c. of gastric contents are employed, and again the computation is based on 100 c.c., the number of cubic centimeters of decinormal sodium hydroxid employed must be multiplied by 20.

If, for example, 2.2 c.c. were required, $2.2 \times 20 = 44 +$ acidity.

Töpfer has shown that alizarin is sensitive for all the elements comprising acidity, except for the combined hydrochloric acid.

The acidity 44, therefore, secured by this reaction must be subtracted from 60, the total acidity; and this gives 16 acidity as the combined hydrochloric acid.

In percentage $16 \times 0.00365 = 0.06$ per cent. combined hydrochloric acid.

The total acidity, free and combined hydrochloric acid, are important to examine for as a matter of routine.

As fractions of a centimeter must often be computed, I give an example in tabulated form:

*Beaker A*, 5 c.c. gastric contents; for *total acidity*, 3.2 c.c. decinormal sodium hydroxid gives end-reaction.

*Beaker B*, 5 c.c. gastric contents; for *alizarin test*, 2.4 c.c. decinormal sodium hydroxid gives end-reaction.

*Beaker C*, 5 c.c. gastric contents; for *free hydrochloric acid*, 1.5 c.c. decinormal sodium hydroxid gives end-reaction.

1. *Total Acidity. Beaker A.*—5 c.c. $\times 20 = 100$ c.c. 3.2 c.c. $\times 20 = 64$ c.c. $64 \times 0.00365 = 0.23$ per cent.

2. *Free Hydrochloric Acid. Beaker C.*—1.5 c.c. $\times 20 = 30$ c.c. $30 \times 0.00365 = 0.11$ per cent.

3. *Combined Hydrochloric Acid* $= A - B$.—

$B =$ alizarin reaction. $A = 64$

$2.4 \times 20 = 48$. $\underline{B = 48}$

$A - B = 16 =$ combined HCl.

$16 \times 0.00365 = 0.06$ per cent.

4. *Total Hydrochloric Acid, Free and Combined.*—0.11 per cent. $+ 0.06$ per cent. $= 0.17$ per cent.

5. *Acid Salts* $= B - C$ *(Organic Acids Absent).*—

$$\begin{array}{r} B = 48 \\ \underline{C = 30} \\ B - C = 18 \end{array}$$

$B - C = 18$

$18 \times 0.00365 = 0.07$ per cent.

If 10 c.c. of gastric juice be employed in the tests, then the number of cubic centimeters of sodium hydroxid required to produce the end-reaction must be multiplied by 10.

If 2 c.c. of gastric juice is used, the multiple is 50, and so on.

The further tests are as follows:

**Lactic Acid.**—*Uffelmann's Test.*—This reagent is the one most frequently employed and is sufficiently accurate, if necessary precautions are observed. It should be freshly prepared before each test. It can be prepared as follows: 10 c.c. of a 4 *per cent. carbolic acid solution* is mixed with 20 *c.c. of distilled water*, and to this is added 1 drop of *sesquichlorid of iron.* A watery solution of carbolic acid (2 per cent.), to which is added 1 drop of liquor ferri sesquichlorid, is another method of preparation. These solutions have an amethyst-blue color.

Other methods of preparation are recommended by Riegel: 20 c.c. of distilled water, 10 c.c. of a 4 per cent. carbolic acid solution, and 0.1 c.c. of neutral 10 per cent. iron chlorid solution; or, dilute the officinal iron chlorid solution with distilled water until the solution is about colorless, and then add a 2 to 4 per cent. solution of carbolic until the amethyst-blue color appears.

A very dilute iron chlorid solution will also give the reaction, but the blue color acts as a contrast.

The lactic acid reaction has been described as a *canary-yellow* or, more often, a *canary-green color* (Fig. 85).

*Fatty acids* produce an *ash-gray* and *inorganic acids decolorize* the blue solution.

As at times the phosphates may be present in the gastric contents and they give the same reaction, a modification has been recommended, which is *practical for general use.*

*Modified Uffelmann.*—Take 5 c c. of the filtrate plus 10 c.c. of ether, and shake in a test-tube for a few minutes; then allow it to stand until the ethereal solution has separated from watery solution. Pour the ethereal part into another test-tube and place it in a glass of hot water to evaporate. Add 1 c.c. of distilled water to the remaining drops, and test for lactic acid with the Uffelmann solution. If the canary color occurs, lactic acid is positively shown.

A larger quantity of the filtrate can be employed with two or three times the quantity of ether. Fleiner does not evaporate the ether, but adds Uffelmann's solution directly to it. The solution will appear yellow at the bottom of the tube if lactic acid be present.

Strauss employs a mixing funnel with two markings, one at 5 c.c. and the second at 25 c.c. (Fig. 87).

Fill to 5 c.c. with the stomach filtrate. Pour on ether (Squibbs') up to 25 c.c. and shake the mixture well. Open the lower stop-cock and allow the fluid to run off until it reaches 5 c.c., and then pour in distilled water to 25 c.c. To this mixture add 2 drops of iron chlorid solution (1 : 9 distilled water), and shake the whole.

Investigations by Strauss show that if one promillimeter of lactic acid be present, an intense green color occurs; if less lactic acid, the color is light green.

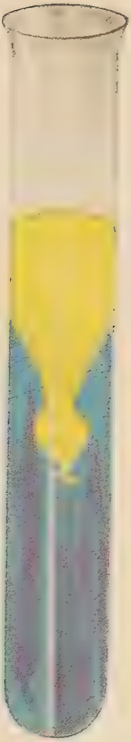


Fig. 85.

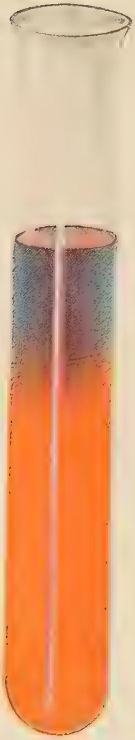


Fig. 86.

Fig. 85.—Uffelmann's test.
Fig. 86.—Congo red test.

Arnold[1] suggests a new test:

1. A solution of gentian violet (0.2 c.c. saturated alcoholic solution in 500 c.c. distilled water), and—

2. Tinctura ferri perchloridi, 5 c.c. (U. S. P.), diluted with distilled water (20 c.c.).

A drop of the iron solution gives a blue color with 1 c.c. of the gentian violet, which changes to yellowish green when gastric contents which contain lactic acid are added.

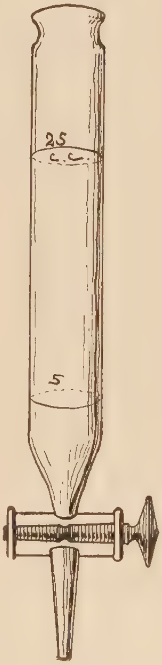


Fig. 87.—Strauss' mixing funnel.

Other methods have been suggested, notably that of Boas, which is rather complicated. It is based on the oxidation of lactic acid into acetaldehyd and formic acid.

The presence of aldehyd is demonstrated by the iodoform reaction with alkaline iodin solution or of aldehyd of mercury with Nessler's reagent.

*Boas' Method.*—Take 10 to 20 c.c. of the gastric filtrate and evaporate it into a syrupy consistency over the water-bath. If free hydrochloric acid is present, an excess of barium carbonate is added. A few drops of phosphoric acid are then mixed in, and the carbonic acid is expelled by boiling. The fluid is then cooled and extracted two or three times with 50 c.c. of ether.

After half an hour pour off the clear ethereal layer. The ether is now evaporated, and the residue washed in a flask with 45 c.c. of water, well shaken and filtered. Concentrated sulphuric acid, 5 c.c. (sp. gr. 1.89), and a pinch of manganese dioxid are added to the filtrate. The mixture is then distilled over a small flame, and the vapor conducted into a narrow cylinder containing 5 to 10 c.c. of an alkaline iodin solution. This consists of equal parts of a decinormal iodin solution and the standard potassium hydroxid solution. The vapor may be conducted into the same quantity of Nessler's reagent. If lactic acid is present, it gives rise to the iodoform reaction (clouding and odor of iodoform) with the iodin mixture. If Nessler's reagent is used, yellowish-red aldehyd of mercury appears.

This procedure is further elaborated for the quantitative estimation of lactic acid, but it is extremely complicated and clinically unnecessary.

We may say that *when a test-breakfast or test-dinner is taken under proper conditions, only traces of lactic acid are introduced, and finding it in appreciable quantities in the gastric contents is of pathologic significance, showing subacidity and stagnation.* It is not pathognomonic of cancer, but these conditions frequently exist in such cases.

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[1] Jour. Amer. Med. Assoc., 1898, vol. viii, p. 21.

*Quantitative Estimation of Lactic Acid.*—The acidity of the filtrate is first determined; 10 c.c. of the filtrate are shaken up with ether in excess. The ether is then separated and the degree of acidity computed therein.

Subtract this figure from the total acidity and multiply by 0.09, which gives the percentage.

Volatile acids must be first tested for and eliminated by boiling. This method is only approximate.

**Volatile Acids.**—Fatty or volatile acids are recognized by boiling a few cubic centimeters of the filtrate in a test-tube. A strip of moistened blue litmus-paper is held over the escaping vapors. The paper will turn red if they are present. Their quantitative determination is hardly necessary.

**Acetic Acid.**—In large quantities acetic acid can be detected by its characteristic odor. For the detection of small quantities, Einhorn neutralizes the watery residue of the ethereal extract of the gastric filtrate with carbonate of soda, and then adds neutral chlorid of iron solution, when a red color is developed.

**Propeptone.**—Add to the gastric filtrate of, say, 5 c.c., an equal quantity of a saturated solution of sodium chlorid. Propeptone, if present, is precipitated. If none is formed, then add 1 or 2 drops of acetic acid, and precipitation will occur if propeptone is present. On heating, the solution clears up, but again becomes turbid on cooling.

**Peptone.**—Preferably, after filtering out the propeptone, take 2 c.c. of the gastric filtrate and make strongly alkaline by adding sodium hydroxid solution and then add a few drops of a weak 1 per cent. copper solution. Peptone gives a purple or violet-red color (biuret reaction).

**Pepsin.**—A thin disk of the white of a hard-boiled egg, weight of about 1 gram (1 cm. in diameter and 1 mm. thick), is placed in a test tube containing 5 c.c. of the gastric filtrate and kept at blood temperature. The tube can be placed in water at blood temperature in a Thermos bottle.

If free hydrochloric acid is not present in the filtrate, add 2 drops of dilute hydrochloric acid. The presence of pepsin will cause disappearance of the albumin in from two to six hours.

The methods for the quantitative determination of pepsin that have been recognized as practical for clinical purposes are those of Hammerschlag and Mett. Henry Illoway[1] has devised a simple method for determining the relative quantity of pepsin, which seems to the author of value: 10 cgm., exact weight, egg-albumen (white of hen's egg) is coagulated in the following manner:

The egg is placed in a small pot of cold water, which is then covered with a lid and put on to boil. It is allowed to cook for ten minutes after the water has begun to boil—in all, heating twenty minutes from the time it has been put on. The egg is then taken out and allowed to cool, either by setting it in a saucer or by putting it in cold water.

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[1] Amer. Jour. Med. Sci., Aug., 1909.

To imitate the usual way, food is, or should be, ingested, the segment of albumin is divided into two parts. Observation has shown, as it has long been known clinically, that thus subdivided the gastric juice can act upon it more quickly. The action being from all sides, it is more effective when we have eight sides for a given quantity than where we have only four.

The coagulated albumin is put into 10 c.c. of the gastric filtrate (from stomach-contents extracted one hour after an Ewald-Boas test-breakfast), and this is then placed in the thermostat, which is kept at 38° C.

The time in which the 10 cgm. are digested, entirely, partially, or not at all, will give us a correct idea as to the status of the pepsin secretion in the case under examination. Illoway, by experiments, shows that normal digestion of the albumin requires from five to five and one-half hours.

He classifies as follows:

*Hyperpepsinia.*—Digestion requiring only from three to four hours, not in any pathologic sense necessarily, but only to indicate a secretion of pepsin greater than usual, which may, however, be the normal for that case.

*Normal Pepsinia.*—Digestion requiring from five to five and one-half hours.

*Hypopepsinia.*—Digestion requiring more than the usual time. The degree indicated by the number of hours required beyond the standard of time.

*Apepsinia.*—No digestion at all.

**Jacoby-Solms Method to Determine Pepsin.**—*Ricin Test.*[1]—Dissolve 1 gram of ricin in 100 c.c. of a 5 per cent. solution of sodium chlorid and filter. Mix 2 c.c. of this filtrate with 0.5 c.c. decinormal HCl solution; 1 c.c. of diluted stomach-contents is added, and allowed to remain at blood temperature for three hours. Ferments clear up the ricin deposit. The quantity of pepsin is determined from the amount of dilution in which the stomach-contents will cause the ricin deposit to disappear.

Solms designates the amount of gastric juice which is sufficient to clear up the 2 c.c. of a 2 per cent. ricin solution in three hours, kept at the blood temperature, as one pepsin unit. The normal stomach-contents contain about 100 pepsin units to the cubic centimeter. Witte[2] has modified the above methods.

Fuld employs a solution of edestin instead of ricin.[3]

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[1] "Ueber eine neue Methode der quantitative Pepsinbestimmung und ihre klinische Verwendung," Zeitschr. f. klin. Med., Bd. 64, Heft 1 und 2.

[2] Berlin. klin. Wochenschr., 1908, p. 643.

[3] "Pepsinbestimmung vermittelst Edestins," Münch. med. Wochenschr., 1907, No. 27, Vereinsbeilage.

An ordinary Thermos bottle partially filled with water at a temperature of about 100° F. can be employed in place of a thermostat, test-tubes containing the solutions being tightly corked and placed therein. Einhorn[1] employs a Thermos bottle with metal framework to hold the tubes. These last are graduated in millimeters, so as to dispense with measuring glasses.

*Casein Test.*—Gross[2] mixes a 1 : 1000 solution of casein containing 16 c.c. of a 25 per cent. HCl to the liter with the filtrate or its dilutions, and leaves it for a quarter of an hour in the thermostat.

He then adds a few drops of the concentrated solution of sodium acetate, which results in a precipitate if the casein has not been digested; otherwise the solution remains clear.

W. C. Rose,[3] has devised a test to be employed as a substitute for the "ricin test," employing a globulin preparation derived from the ordinary garden pea (*Pisum sativum*).

*Mett's Method.*—This consists in sucking fresh egg-albumen into capillary tubes of 1 or 2 mm. diameter, coagulating the albumin by boiling, and then cutting off portions 3 to 5 cm. long of the filled tube and adding these pieces to the gastric contents. This should be kept at body temperature for ten hours in the incubator. At the end of this period each end of the tube will show a lack of solid albumin, owing to digestion, while some will remain in the central portion. Both the empty portions and the portion that is full are measured, and the activity of the pepsin digestion is thus determined. The relative amount of pepsin varies according to the square of the length of the empty portion of the tube, the figures of the latter being expressed in millimeters; thus, 3 mm. of digestion equals 9 parts of pepsin; 2 mm., 4 parts of pepsin, etc.

**Rennet.**—Add 5 drops of the filtered gastric contents, preferably neutralized with decinormal sodium hydroxid solution, to 10 c.c. of fresh neutral milk in a test-tube. Place this in a glass of warm water at a temperature of about 100° F., or in a thermostat. A Thermos bottle containing warm water is extremely convenient.

*Normal Rennet.*—In about five to fifteen minutes coagulation will occur if the rennet be normal. If the same quantity of filtrate (5 drops) be added to 20 c.c. of milk, Illoway finds, under normal conditions, coagulation will occur within fifteen to thirty minutes. He suggests a simple quantitative method.

*Deficient Rennet.*—Add 1 c.c. of gastric filtrate to 10 c.c. and 20 c.c. of milk if the smaller quantity of filtrate (5 drops) gives no result. If reaction occurs within the same period, rennet is deficient.

*More marked deficiency* when no result is obtained with 1 c.c. of gastric filtrate, but is obtained within the half-hour with 5 c.c. of filtrate.

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[1] Einhorn, "A Simplification of the Jacoby-Solms Ricin Method for Pepsin Determination," Med. Record, Aug. 29, 1908.

[2] "Die Wirksamkeit des Pepsins und eine einfache Methode zu ihrer Bestimmung," Berlin. klin. Wochenschr., 1908, No. 13, p. 643.

[3] Arch. of Internal Medicine, May, 1910, vol. 5, pp. 459–465.

*Absence of Rennet.*—When no reaction is obtained within half an hour with 5 c.c. of gastric juice and 10 c.c. of filtrate, no reaction will occur.

It has been demonstrated by Illoway's experiments that rennet may be present in nearly normal amount, even if pepsin is markedly deficient; it may be present even if pepsin is absent.

Rennet is usually the last one of the elements active in the process of gastric digestion to disappear.

*Rennet Zymogen (Chymosinogen).*—Add to the same specimen of milk 3 to 5 drops of a 1 per cent. solution of calcium chlorid and place in an incubator. Coagulation shows the presence of the enzyme; otherwise it is absent.

**Examination of Starch Digestion.**—The salivary ferment continues its action on starch in the stomach while the amount of hydrochloric acid is not too great. It is estimated that as soon as the total hydrochloric acid reaches 0.12, the action ceases. If the secretion of hydrochloric acid is abnormally great, starch digestion is soon stopped, and there is either no digestion of starch or the end-products are not formed. The reverse is the case in subacidity.

To determine the intermediary stages, a dilute iodin-potassium solution (Lugol's) is employed. It consists of iodin, 0.1; potassium iodid, 0.2; aqua destillata, 200.0.

A few drops of the filtered gastric juice are placed on a porcelain dish, and to it is added a drop or two of Lugol's solution. The reactions are as follows:

Dextrin turns the fluid blue; erythrodextrin, a red purple; achroödextrin discolors slightly the yellow tincture of the Lugol; maltose does not change the color.

For sugar or maltose, Fehling's or Trommer's test must be employed.

In normal cases, sugar and achroödextrin are present; erythrodextrin absent or present in small amount; dextrin absent.

If blue or blue-violet color appear, saccharification is deficient.

**Other Methods for Determining Free Hydrochloric Acid.**—It seems advisable to describe a few additional practical methods of determining free hydrochloric acid:

1. *Tropäolin* 00 (Merck's) in a concentrated watery solution is recommended by Riegel. Knapp employs a supersaturated alcoholic solution of the same.

To 5 c.c. of the filtered chyme add 2 drops of the tropäolin solution. Free hydrochloric acid turns it a cherry-red. Titrate with decinormal sodium hydroxid until it becomes *amber*—the end-reaction.

As 5 c.c. is $\frac{1}{20}$ of 100, on which the calculation is based, and if it takes 3 c.c. of the alkali to produce the end-reaction, free hydrochloric acid therefore $=20\times3=60$.

Multiply $60\times0.00365$ to secure percentage.

2. *Mintz's Method.*—For example, to 10 cc. of the gastric filtrate, decinormal sodium hydroxid is gradually added, until 1 drop of the mixture no longer responds to the Günzburg reaction (phloroglucin-vanillin test). A platinum loop should be employed as a drop carrier.

The amount of the sodium hydroxid solution in this case should be multiplied by 10 to give free hydrochloric acid.

For example, if the reaction no longer appears after the addition of 3 c.c. sodium hydroxid, free hydrochloric acid = 30; percentage = 30 × 0.00365, or 0.109 per cent.

3. *Boas and Moerner.*—They estimate the free hydrochloric acid by Congo paper, or by a 1 per cent. watery solution of Congo red, which turns blue in the presence of this acid (see reaction, Fig. 86).

Take 5 c.c. of the filtrate and add 3 or 4 drops of the Congo red solution. More is unnecessary, though Boas adds 5 c.c. of it.

On the other hand, Congo paper can be moistened in the filtrate. A blue reaction results in each case.

Decinormal sodium hydroxid is then added to the mixture, or the paper is placed in a porcelain dish and the alkali added. Titration is continued until the blue begins to turn red. The estimation is performed in the same way as before.

4. *Riegel's Method.*—Congo paper is employed in his test.

Take 10 c.c. of the gastric filtrate. The Congo paper is dipped in the filtrate, giving the blue reaction of free hydrochloric acid. It is then placed on a saucer. Decinormal sodium hydroxid is allowed to drop slowly from the buret into the filtrate, and a drop of the mixture is removed from time to time with the platinum loop and applied to the Congo paper. As the change in color becomes indistinct, this is controlled by dropping distilled water on the same piece of paper. The alkali is dropped on the paper until it begins to turn a violet red.

The number of cubic centimeters of decinormal sodium hydroxid necessary to secure the end-reaction, say, 3 c.c., is multiplied by 10, giving free hydrochloric acid as 30. This is necessary, as the original figure is computed for 100 c.c. of contents, and only 10 c.c. were employed.

To complete the analysis and estimate the total acidity, 2 drops of phenolphthalein solution are added to the same filtrate, and titration with the sodium hydroxid is continued until the color of the solution turns red (the end-reaction).

The total acidity is indicated by the total quantity of decinormal sodium hydroxid used from the beginning of the first titration. For example, if in both titrations 6 c.c. of sodium hydroxid have been employed, the total acidity is 60, as 10 c.c. of filtrate were examined.

Various modifications have been employed for these tests, which serve only to confuse the reader. Those described are the most practical.

Small booklets of Congo paper can be secured from Merck. It may be prepared by saturating filter-paper with a watery solution. It is of a reddish-pink color.

The qualitative examination for free hydrochloric acid can be made with this paper by dipping it into the filtered or unfiltered gastric contents. If free hydrochloric acid is present, it will turn blue.

**Determination of Hydrochloric Acid Deficit.**—Honigmann[1] and von Noorden[2] determine the degree of hydrochloric acid insufficiency by adding a decinormal hydrochloric acid solution to the stomach-contents until free hydrochloric acid can be detected by Congo paper or Günzburg's test.

Ten c.c. of the filtrate are placed in a beaker and the decinormal hydrochloric acid solution allowed to flow into it gradually, the solution being well mixed. The test being continued until after repeatedly dipping the Congo paper into it, the paper shows a bluish tinge.

The more hydrochloric acid required to secure the reaction, the less the amount of combined acid in the filtrate.

Or, about 25 drops of Günzburg's solution can be added to the filtrate, and then titration with the dilute acid follows until a red mirror appears on a porcelain dish as a couple of drops of this mixture are evaporated over an alcohol-lamp.

We know, however, that the average amount of combined hydrochloric acid is 25, or 0.1 per cent., under normal conditions, and *Töpfer's method of testing will give the required data.*

The amount of peptone and propeptone qualitatively are an indication. When there is no biuret reaction, there will be no combined hydrochloric acid.

**Other Methods of Testing the Gastric Secretion.**—There are several ingenious methods for testing the gastric secretion in order to obviate the unpleasant procedure of aspiration, or because some patients absolutely refuse the tube, or there is danger incurred from its passage, such as in cases of aneurysm, angina, severe endocarditis, or after a recent hemorrhage from the stomach or lungs, or in the very debilitated.

*Sahli's Desmoid Test.*[3]—This consists in placing methylene-blue or iodoform in a small rubber bag and tying it tightly with thin raw catgut. The bag is swallowed after a large meal, and the urine is examined for methylene-blue, or the saliva for iodin. Methylene-blue colors the urine green or greenish blue. The iodin is tested for in the saliva by starch-paper and fuming nitric acid, giving a bluish or violet color. It is based on the fact that raw connective tissue, including catgut, is, according to Schmidt,[4] digested only by the gastric juice and not by the pancreas.

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[1] Berlin. klin. Wochenschr., 1893, Nos. 15 and 16.

[2] Ibid., No. 19.

[3] Correspondenzblatt für Schweizer Aerzte, 1905, Nos. 8 and 9.

[4] Deutsch. med. Wochenschr., 1899, No. 49.

The reaction occurs in healthy persons usually six to eight hours after swallowing the bag. If it takes place later, or not at all, the secretory function is insufficient. An early reaction shows hyperacidity, according to Kaliski.

Einhorn,[1] in some cases of achylia gastrica and cancer, has demonstrated that catgut is also digested in the bowel, and considers that the method is unsuitable.

2. *Günzburg's Method.*[2]—This is based on the same principle as Sahli's. The patient swallows 0.2 gm. potassium iodid, inclosed in a small rubber bag, which is tied with fibrin threads.

After the fibrin is digested, the potassium iodid is set free and absorbed. The test of the saliva is made with starch-paper and fuming nitric acid, giving a violet or bluish color. This necessitates frequent examinations of the saliva, and the bag may escape and the fibrin be digested in the intestine. The objections are the same as to Sahli's test.

3. *Spallanzani and Edinger's Method.*[3]—They fasten a small sponge to a silk thread. The patient swallows this, and after several minutes it is withdrawn and the contents squeezed out and examined. Some of the contents are squeezed out during removal, and secretions of the esophagus and pharynx are mixed with it.

4. *Dunham's Thread Test.*[4]—The patient swallows a small tassel of test thread, colored with litmus, Congo, or dimethylamido-azobenzol, attached to a silk thread about 30 inches long. After about five minutes it is withdrawn and the color reaction noted. There are the same objections as to the previous test.

5. *Einhorn's Stomach Bucket.*[5]—This consists of a small silver capsule, open at the top, with a cross-piece as a handle, to which a silk thread is attached. Several sizes can be secured (Figs. 88 and 89).

The bucket is moistened in warm water and emptied. It is then placed on the root of the tongue and swallowed by the patient. The vessel should remain in the stomach for about five minutes. It is then withdrawn by the silk thread.

During the act of withdrawal, if resistance is felt, the patient is told to swallow and the bucket is then readily removed.

If there are mucus and saliva in the patient's mouth, he should first gargle with plain water and expectorate the mucus.

The test-breakfast should be given and the bucket administered an hour later. The gastric contents are poured from this instrument into a small beaker. Tests can then be made with blue litmus and Congo paper. A single drop can be tested with Günzburg's solution.

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[1] Jour. Amer. Med. Assoc., May 12, 1906.

[2] Deutsch. med. Wochenschr., 1889, No. 4.

[3] Deutsch. Arch. f. klin. Med., vol. xxviii, 1881.

[4] New York University Bulletin of the Medical Sciences, vol. 1, No. 4, p. 178, Oct., 1901.

[5] Medical Record, July, 1890.

Einhorn demonstrates that the amount of hydrochloric acid can be approximately determined by diluting 1 drop of the filtrate with water, until Günzburg's reaction begins to disappear in the diluted fluid. He finds that normally the drop can be diluted up to eight or ten times and give the Günzburg reaction. The disappearance of the reaction after five times dilution or less shows subacidity.

The presence of the reaction after dilution of twelve times or more shows hyperacidity.

Recently he has employed another approximate method with a strip of paper saturated with 0.5 per cent. dimethyl-amido-azobenzol solution and with dilution. If the paper turns red after dilution of 1 drop of filtrate with 3 to 6 drops of water, free hydrochloric acid is normal; if no color appears after 3 drops of dilution, it is subacid; and if red appears after over 6 drops dilution, it is hyperacid.

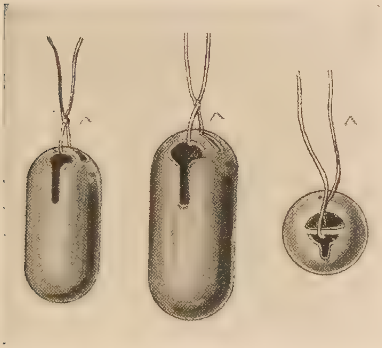

Fig. 88.—Einhorn's stomach bucket, three sizes.

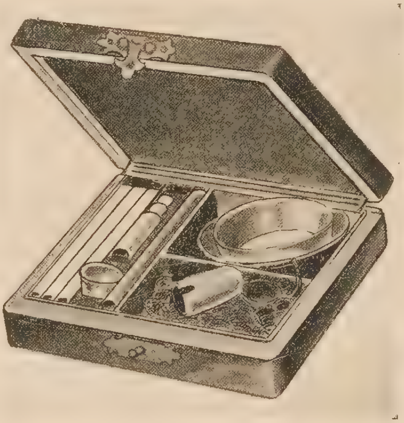

Fig. 89.—Stomach bucket set.

If rennet is present, pepsin is also present; 2 drops of filtrate added to 2 c.c. of milk placed at blood temperature in a tube in warm water will curdle in ten to twenty minutes in the presence of rennet.

I employ this method in exceptional cases, when the stomach-tube cannot be passed. It is *preferable to aspirate when possible.*

**Gas Fermentation.**—The presence of free hydrochloric acid does not prevent the development of gas. It may occur in a soil that is non-acid, together with lactic acid fermentation. Stagnation is the chief factor favoring fermentation. Various gases may be formed, but from a practical point of view the determination of its occurrence, quantity, and the time necessary for its development are sufficient.

The unfiltered gastric contents should fill a tube such as is employed for the determination of sugar in the urine (the Fiebig tube). It should be placed in an incubator at 37° C. (98.6° F.), or if this is not at hand, in a uniform warm place. One can employ a test-tube, as suggested by Moritz, closed by a rubber cork through which is passed a bent glass tube. The test-tube is filled with gastric contents and then closed with the cork, thus forcing some of them into the curved tube and preventing the entrance of air. The apparatus is inverted in a beaker.

As at times the sugar may already be destroyed by fermentation, it is well to prepare a control tube of gastric contents to which a small quantity of powdered dextrose has been added, or it may be added at once. If no development of gas is noted after twenty-four hours, the tube should be allowed to stand for three or four days.

Carbon dioxid may be identified by allowing a small amount of KOH to flow through a pipet to the bottom of the gas column. The carbonic acid is absorbed by it and the fluid moves up to take the place of the absorbed gas. The expressed test-meal, or test-breakfast, or vomitus can be employed.

If fermentation is excessive, one can assume motor insufficiency. If rapid fermentation occurs within a few hours, pyloric stenosis should be suspected, as this produces the most severe degree of motor insufficiency.

If the stomach contains a large amount of lactic acid and no free hydrochloric acid, carcinoma of the pylorus is probably the cause of this stenosis.

Gaseous fermentation is usually more intense in cases of motor insufficiency in which free hydrochloric acid is present. It can occur, however, in any form of stomach disease in which there is a disturbance of gastric secretion. Lactic acid fermentation only occurs markedly in subacid conditions. Alcohol, various hydrocarbons, and sulphureted hydrogen have been found as products of fermentation. Boas finds $H_2S$ chiefly in benign ectasia.

**Determination of the Quantity of Chyme Within the Stomach.**—Usually the quantity of chyme can be determined by emptying the patient's stomach by the expression method, especially with the addition of the aspirating bulb that I have described.

Air can be forced into the stomach by covering the free end of the bulb and squeezing the latter. If no bubbling sound is heard, the organ may be considered empty. With marked dilatation from pyloric stenosis, it is at times difficult completely to empty the organ.

Mathieu and Rémond suggest the following method of determining the total quantity:

After removal of the contents a funnel is attached to the tube and 200 c.c. of water poured into the stomach. The funnel is moved up and down several times, and the patient shakes the abdomen so that complete mixture of the water and contents occurs. They are then removed by a combination of siphonage and expression.

The quantity of liquid originally contained in the stomach is equal to the number of cubic centimeters of water poured into the stomach multiplied by the degree of acidity of the second portion removed, divided by the figure resulting by deducting the degree of acidity of the second portion from that of the first plus the portion previously withdrawn.

**Examination of the Vomit.**—The same methods that are employed in examination after the test-meal apply to investigation of the vomitus. The information, however, is not as positive, as the admixture of bile, saliva, etc., obscures the result. Important information is secured, but the physician should inspect the vomitus in person.

If the vomiting consists chiefly of food, it should be learned when the last meal was taken and of what it consisted. If coarse morsels are found six or seven hours after a meal, the conclusions are naturally different than if they were found directly after eating; and by this means we can often determine the digestive and motor powers of the patient.

If food is vomited that was ingested the day before, marked motor insufficiency is present.

With gastric ulcer, vomiting often occurs at the height of digestion. In some nervous diseases it usually takes place immediately after eating. With ectasia, vomiting occurs late in digestion and often in great quantity.

In dilatation with hypersecretion the fluid is abundant, and there are fine remnants of amylaceous material; while with carcinoma of the pylorus undigested morsels of meat are present. In the former case free hydrochloric acid is present, and Congo paper will turn blue; in the latter case, no color change occurs.

The presence of blood is important, and its appearance depends on the presence or absence of hydrochloric acid, on the rapidity with which it is poured forth, and on the length of time it has remained in the stomach. It may, therefore, appear chocolate brown, like coffee-grounds, or as fresh blood.

Mucus is also readily discovered. Pus is rarely found, unless from perforation in phlegmonous gastritis, or from some neighboring focus into the stomach, or unless it has been swallowed. *Microscopic pus occurs with ulceration.*

During violent vomiting bile is frequently in evidence, or when vomiting occurs on an empty stomach. It may occur with pyloric stenosis, when the opening is kept slightly patent. With persistent vomiting of bile one would suspect some obstruction of the duodenum, such as carcinoma, torsion, gall-stones, etc.

Parasites, such as ascarides or the oxyuris vermicularis, are occasionally found in the vomit, and very rarely a piece of gastric tumor.

**Examination of the Contents of the Fasting Stomach.**—It may be necessary to investigate the contents of the fasting stomach, especially when disturbance of the motor function is present or when hypersecretion is suspected. Under normal conditions one might expect to find from 5 to even 15 c.c. of gastric contents in the normal stomach. Anything over 20 c.c. is considered pathologic.

The examination should be made in all cases where one suspects an abnormal quantity of gastric contents will be found in the morning after fasting; also in all cases of suspected ectasia or atony of the stomach.

The best method is to wash the stomach thoroughly the night before, both in the sitting and reclining posture, and then administer a test-supper. The contents are aspirated, measured, and examined the following morning before breakfast. The chief purpose is to test the motor function of the organ, especially to determine whether motor insufficiency of a high degree is present.

When hypersecretion is suspected, the procedure is slightly different. The stomach is thoroughly washed at about 10 P. M. and care taken that all the water is removed. No food or drink is allowed thereafter, and in the morning before breakfast the contents are aspirated and examined. A quantity over 20 c.c. found repeatedly I would consider pathologic (hypersecretion).

When there is permanent regurgitation of bile into the stomach, Riegel has shown that this method of examination is important. If bile and pancreatic juice enter the stomach, digestive processes are arrested.

Bile, pancreatic, and probably intestinal juice are occasionally found in the empty stomach (duodenal juice, Boas). This material is grass green or yellowish, containing bile constituents, and converts starch into maltose and dextrose, proteins into peptones, and splits fats. If it is aspirated occasionally, it is probably not significant; but if there is constant regurgitation, it is suggestive of obstruction in the duodenum.

When there is obstruction to the flow of the intestinal contents, or a communication between the stomach and intestine, intestinal contents are found in the stomach.

## ABNORMAL CONSTITUENTS OF THE STOMACH-CONTENTS

Abnormal products which are of importance for our diagnosis are quite frequently found in the gastric contents. They may contain mucus, blood, bile, intestinal juice, and pus.

*Mucus when present in considerable quantity is easily recognized.* It generally occupies the upper part of the fluid, appearing in swollen, glassy lumps or in flakes and shreds. It is also intimately mixed with the food. It can be readily lifted with a glass rod. If in small amount, a few drops of dilute acetic acid are added, it will be revealed by the characteristic precipitate.

**Bile and Intestinal Juice.**—Small quantities of bile and intestinal juice may occasionally be met with normally. In the paragraph on "Examination of the Contents of the Stomach after Fasting" I referred to the presence of bile and intestinal juice, and that their frequent occurrence is due either to relaxation of the pylorus or stenosis of the duodenum below the mouth of the bile-duct. Pure bile is golden yellow, but green if mixed with gastric juice. I believe that too often the diagnosis is made by simple inspection. Mold may produce a greenish color, and I have found it on several occasions. For accuracy, the tests should be made—Gmelin's for bile-pigment, and Pettenkofer's for bile-acids.

Einhorn suggests the following for the intestinal juice, which is recognized by its ferments, trypsin, amylopsin, and steapsin:

**Trypsin.**—Mix the filtrate with 1 per cent. solution of sodium carbonate until the reaction is decidedly alkaline; add a flake of fibrin and keep in a warm place for several hours. Trypsin will dissolve the fibrin.

**Amylopsin.**—Starch is changed into maltose.

**Steapsin.**—Add 1 drop of blue litmus tincture and a few cubic centimeters of the neutralized filtrate to a small portion of milk and keep at blood temperature. Steapsin changes the blue color, and the milk becomes slightly reddish from decomposition of the fat into fatty acids.

**Blood.**—Blood in large quantities is easily recognized, as is fresh blood, even if in small amounts.

Fresh blood mixed with gastric contents presents a reddish appearance, while old blood is brownish or of a coffee-ground color. It may even appear blackish. When the blood cannot be detected microscopically and gastric hemorrhage is suspected, *occult blood* must be examined for.

It is advisable to make the same *examination of the stool.*

1. **Benzidin Blood Test for Gastric Contents and Stool.**—This is a test devised by O. and R. Adler.[1] They first applied it to test the feces. Schlesinger and Holst[2] advise boiling the gastric filtrate and testing in the same manner as for feces.

*Gastric Contents.—Solution* 1.—Knifepointful of benzidin (Merck's) is added to 2 c.c. of glacial acetic acid and allowed to stand and dissolve.

*Solution* 2.—Ten to 12 drops of the benzidin solution are added to 2½ or 3 c.c. of a 3 per cent. peroxid of hydrogen solution.

Three or 4 drops of the *gastric filtrate,* which has been boiled for about half a minute, are added to Solution 2. In the presence of blood, a green or blue color results in from a few seconds to a minute.

*Stool.—The stool should also be examined.* A small piece of feces about the size of a pea is mixed with 2 c.c. of water and boiled in a test-tube closed with cotton for half a minute; 3 or 4 drops of the boiled fecal solution are added to Solution 2. A green or blue color results if blood be present.

For making this test Paul Cohnheim has devised a slight modification. Place a little benzidin (Merck) in a dry test-tube and shake it with about ½ c.c. of glacial acetic acid; add 2 c.c. of $H_2O_2$ and then carefully place on its surface a little of the fluid which is to be examined.

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[1] Zeitschr. für physiol. Chemie, Bd. 41, Heft 1 and 2, p. 59.

[2] Deutsch. med. Wochenschr., 1906, No. 36, p. 1444.

The last should be previously boiled. A green-tinted ring results. The intensity of the green or bluish ring affords one conclusions as to the quality of the occult blood.

*Benzidin Test Paper.*—Einhorn[1] has devised a benzidin testing paper as follows:

Take a saturated solution of benzidin and glacial acetic acid; moisten filter-paper therein and dry it. Both in preparing the paper and making the test avoid contact with the fingers, as a drop of perspiration causes the reaction. When handling the paper, employ ivory-tipped forceps or protect the hand by a towel.

*Method.—Gastric Contents.*—A piece of benzidin paper is first immersed in the gastric filtrate, and then a few drops of hydrogen peroxid are added. The paper is then placed on a piece of white porcelain and examined for the development of a blue color. If blood is present, a blue or green color occurs in a *few seconds to a minute.* Einhorn shows that if we wait longer periods, other substances may cause the reaction, also in time the paper moistened with peroxid will become blue.

*Feces.*—In testing for occult feces with the paper, a small piece the size of a pea is rubbed up with 2 c.c. of water, the benzidin paper immersed therein, a drop of hydrogen peroxid added, and the blue color then examined for.

The benzidin paper Einhorn recommends as a preliminary, and if there is immediately a strong reaction or none at all, he regards the result as reliable. If at the end of a minute only a trace of reaction occurs, then the aloin-ether extract method may be employed as a check.

In the fecal examination, ether extract of feces, as employed in the aloin test, makes the benzidin test more reliable.

In *examination of the gastric contents* no meat products or iron preparations should be taken *for at least twenty-four hours before the test,* and in testing the stool *the same rule must be observed;* but, preferably, *for a longer period if possible*—at least two to three days as a precautionary measure.

2. **Phenolphthalein Test for Occult Gastro-intestinal Hemorrhage.**—The *reagent*[2] is made as follows: Dissolve 1 gm. of phenolphthalein and 25 gm. of potassium hydroxid in 100 c.c. of water, and reducing with 10 gm. of pulverized zinc. The resulting red fluid is stirred or shaken over a small flame until it is entirely decolored, the phenolphthalein being reduced to phenolphthalin. The solution is then filtered, when it is ready for use. It keeps indefinitely.

A small amount of the stool, about 5 c.c., is rubbed up in water until it forms a thin fluid. Into this a little glacial acetic acid is stirred to acidify, ether is then added in equal volume, and the containing glass is slowly moved to and fro until the contents are well mixed. The ethereal solution is then decanted into another reagent glass and 20 drops of the phenolphthalin reagent are added. This last is shaken, and then 3 or 4 drops of hydrogen dioxid are added. In the presence of blood the phenolphthalin is oxidized into phenolphthalein, and as it is in alkaline medium, the fluid *turns pink.* With considerable blood the *pink tint persists* for some time; with little blood, it soon fades. With much blood, the reaction is pronounced, even without the hydrogen dioxid. To test stomach-contents, a few drops of glacial acetic acid are added to a few cubic centimeters of gastric filtrate and the same methods followed.

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[1] "A New Blood Test," Med. Record, June 8, 1907.

[2] Boas believes this method the best. Deutsche med. Wochenschr., Berlin, Jan. 12, 1911, vol. xxxvii, No. 2.

3. **Weber's[1] Modification of Van Deen's Test for Occult Blood.**—Dilute the *stomach-contents* or, preferably, the *filtrate thereof*, with $\frac{1}{3}$ volume of glacial acetic acid, and extract with about 10 c.c. of ether. A few cubic centimeters of this acid ether extract are mixed with 10 drops of tincture of guaiac and 20 to 30 drops of ozonized oil of turpentine (old turpentine exposed to air).

If blood is present, the mixture turns a blue or blue violet; if absent, it turns a reddish brown with a green tinge. The reaction is more distinct if a little water is added and the blue pigment extracted with chloroform.

Many authors insist that the tincture of guaiac should be freshly prepared on each occasion. Soper[2] demonstrates that this is unnecessary. He reduces the guaiac resin in a mortar to a fine powder, slowly adding 95 per cent. alcohol, leaving a residuum of guaiac in the mortar to insure a strong tincture. This is filtered and kept in a glass-stoppered bottle as a stock preparation. Dilute a portion of this stock tincture with 95 per cent. alcohol (tincture, 1 part; alcohol, 5 parts), and keep in a smaller glass-stoppered bottle for daily use.

*The Stool.*—The test for occult blood is as follows:

Treat 5 c.c. of feces with 20 c.c. of ether; the latter is then poured off; 2 c.c. of glacial acetic acid are added to the feces and thoroughly stirred. This mixture is again treated with about 10 c.c. of ether and allowed to separate.

To 2 c.c. of the etherized extract add 2 or 3 drops of a fresh tincture of guaiac. Then add 20 to 30 drops of ozonized oil of turpentine, or pure hydrogen dioxid, and shake well. If blood be present, there appears a blue or blue-violet color.

*Meat and iron preparations should be avoided for from twenty-four to seventy-two hours before these tests.*

4. **Klünge's Aloin Test.**—*Feces.*—In this test freshly prepared aloin is employed. Dissolve as much aloin as can be placed on the tip of a knife-blade in 10 c.c. of 70 per cent. alcohol; add 2 c.c. of the aloin solution to 2 c.c. of the ethereal extract of feces, prepared as above, and then the oil of turpentine or dioxid of hydrogen as described. A cherry-red color appears in the fluid if blood be present.

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[1] Berl. klin. Wochenschr., 1893, No. 19.

[2] Jour. Amer. Med. Assoc., Jan. 28, 1911.

*Gastric Contents.*—Ethereal extract of the filtrate is prepared as in Weber's test. The rest of the test is the same as the aloin test of feces.

Einhorn at times employs aloin paper prepared with filter-paper saturated with a solution of aloin in 70 per cent. alcohol, the paper being then dried for future use. I prefer the *benzidin* or *Weber's test.*

The spectroscopic test—Heller's, Schönbein's, Korcznski's, the hemin test, etc.—have been suggested, but the ones described are the most practical.

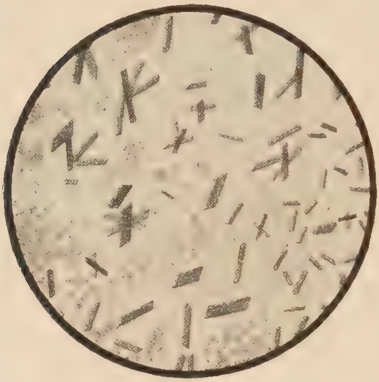

Fig. 90.—Teichmann's hemin crystals (Jakob).

*Hemin Crystals.* — Hematin combines with one molecule of hydrochloric acid to form hemin. This last substance crystallizes in brown plates or columns, and when produced by the addition of glacial acetic acid, may be of considerable size. Star or rosette-shaped crystals may also be present. The formation of hemin may occur from mere traces of blood. Negative results are not conclusive.

The test is as follows: Evaporate a small sample of gastric filtrate on a watch-crystal over a small alcohol flame. Scratch the residue free, and mix with it a grain or two of finely powdered salt. Transfer this to a microscopic slide, and add a drop or two of glacial acetic acid. Gently heat the slide for a minute or two until bubbles begin to form, then cool it off and examine under the microscope with a one sixth or one-seventh objective. The crystals will be seen as in Fig. 90.

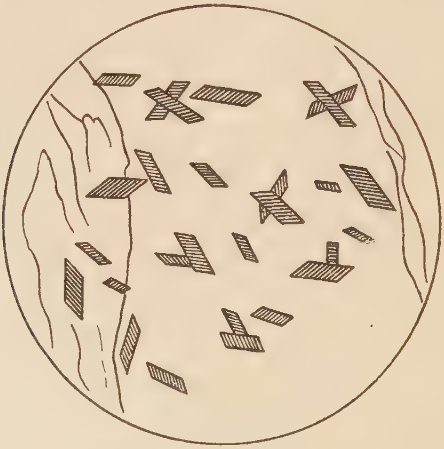

Fig. 91.—Crystals of hydriodic hematin ester (Gross).

Gross[1] employs Stozyzowski's modification of the above (Teichmann's)[2] method. He recommends it particularly in the determination of blood in suspected duodenal ulcer, first obtaining the duodenal contents by his aspirator. He notes small apparent blood points in the contents, and placing one of these on a slide, drying it slightly, then covers it with a cover-glass. At the margin he places one or two drops of the following mixture: alcohol, water, glacial acetic acid, of each, 1 c.c., and 3 drops of hydriodic acid (undecomposed, if possible, and of a specific gravity of 1.5). The specimen surrounded by this solution is boiled for about ten seconds over a small spirit lamp. The loss sustained by evaporation should be constantly replaced. Objective 7, ocular 3, should be employed in the microscope. If the specimen is blood, there will be rhomboid prismatic crystals of a black color (hydriodic hematin ester), as in Fig. 91.

Pus is seldom found in the gastric contents, and is recognized readily under the microscope. Excluding ingested pus and phlegmonous gastritis, pus shows ulceration of the gastric mucosa.

## MICROSCOPIC EXAMINATION OF THE GASTRIC CONTENTS

The relative value of the microscopic examination of the gastric contents after the test-meal, of the vomitus, and of the fasting stomach-contents, as compared with gastric analysis, is still a matter of dispute.

Some of the ardent advocates of the microscope go so far as to claim that their method is alone necessary. Undoubtedly, in many cases the clinical symptoms, gastric analysis, the test of the motor function, and macroscopic inspection of the contents afford sufficient information for diagnosis. I do not wish, however, to depreciate the value of the microscope, as in some cases it is of fundamental importance.

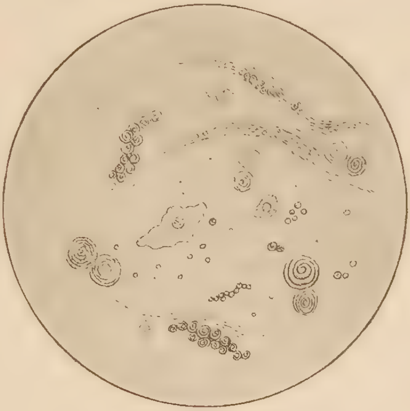

Fig. 92.—Fasting gastric juice containing mucus, snail forms, epithelial cells, and amorphous material.

**Gastric Secretion.**—When fasting, the gastric secretion shows normally under the microscope epithelial cells, cell nuclei, some mucus, amorphous material, and micro-organisms. Jaworski[3] describes spiral or snail-like bodies in cases of hyperchlorhydria, but Boas believes they are quite common and that they are developed from the mucus by the action of the gastric juice (Fig. 92). Einhorn has found them in patients with normal secretion.

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[1] New York Med. Record, April 22, 1911.
[2] Therap. Monatsh., Sept., 1902.
[3] Münch. med. Wochenschr., 1887, No. 30.

**Mucus.**—Mucus from the bronchi and lungs is characterized by the presence of alveolar cells and myelin drops; while the occurrence of a great many columnar epithelial cells is evidence of its origin from the gastric mucous membrane.

In *doubtful cases* the microscope will thus determine the source of the mucus, either by examination of the fasting contents or after a test-meal. The clinical symptoms and macroscopic appearance of the contents, as described under Chronic Gastritis, will, however, generally give sufficient information.

Paul Cohnheim holds that the presence of free nuclei of leukocytes and epithelial cells is a positive evidence of hydrochloric acid and pepsin (Fig. 93).

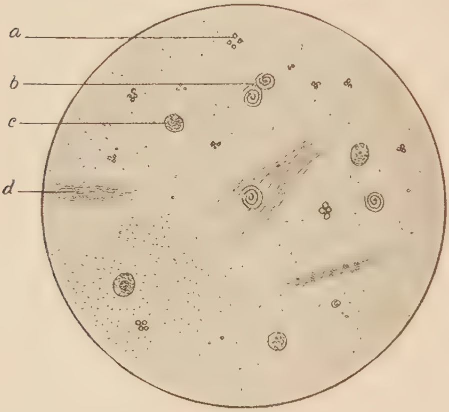

Fig. 93.—*a*, Nuclei of leukocytes; *b*, spiral bodies; *c*, nuclei of epithelial cells; *d*, striated mucus.

I believe that gastric analysis is much preferable for such determination. Importance has been attached to the presence of two varieties of infusoria, the Trichomonas hominis and Megastoma entericum, notably by Cohnheim (Fig. 94). He believes that *their presence is pathognomonic of carcinoma*, not *affecting the motility of the stomach.* Amebæ are often associated with them.

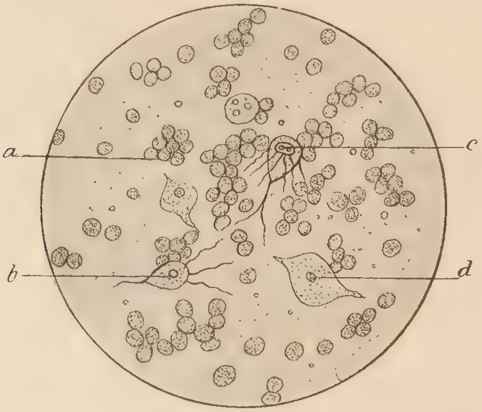

Fig. 94.—*a*, Pus cells; *b*, trichomonas; *c*, megastoma; *d*, pavement epithelium.

The development of these infusoria requires the absence of hydrochloric acid, an alkaline medium, and the existence of deep folds in the mucosa. Previous to aspiration, the stomach-tube and receptacle should be warmed.

To differentiate between cancer and achylia gastrica in the suspected cases with emaciation, gastric distress, and achylia, special examination for these infusoria is advocated by Cohnheim. The presence of pus, especially if associated with blood in the nonfetid gastric contents, he also believes aids in the early diagnosis before the tumor is palpable. Microscopic pus in the gastric contents shows ulceration, but not necessarily malignant in type. Phlegmonous gastritis and the ingestion of pus from above must be excluded.

Sarcinæ and the Boas-Oppler bacillus can be examined for, both in the fasting stomach and after the test-meal. The same is true as regards epithelial cells and pieces of the gastric mucosa. I will refer to them shortly.

**Gastric Contents.**—The microscopic examination of the gastric contents after the test-breakfast or dinner shows, under normal conditions, a few starch granules, many of which no longer appear in spiral form. The muscular fibers do not show their diagonal stripes; globules of fat, plant-cells, and micro-organisms are present in small numbers.

Many unchanged starch granules are found in hyperchlorhydria and hypersecretion, and the muscle-fibers are well digested; while with hypochlorhydria (deficient secretion) unchanged muscle-fibers are present. The granules of starch are brought out clearly by the addition of one drop of tincture of iodin, giving a blue reaction. The microscopic findings are here confirmatory of macroscopic inspection.

The varieties of micro-organisms have been thoroughly studied by DeBary,[1] Nencki,[2] Boas,[3] and others. It has been demonstrated that they may be present even in hyperchlorhydria, showing that the hydrochloric acid does not always prevent fermentation.

J. Kaufmann[4] has described a case of hyperchlorhydria in which the motor function of the stomach was not markedly disturbed, but which showed fermentative processes. He isolated eight varieties of micro-organisms in a specimen of the gastric contents.

Boas has also described the development of sulphureted hydrogen in a case of hyperchlorhydria. In general, we may say that *fermentation develops in cases when the motility of the stomach is reduced.*

Minkowski[5] has shown that if free hydrochloric acid be abundant, yeast and thread fungi may be found; while if it is absent, numerous

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[1] Archiv. f. exper. Path. und Therap., Bd. 20, p. 243.

[2] Archiv. f. exper. Path., Bd. 28.

[3] Deutsch. med. Wochenschr., 1892.

[4] Berlin. klin. Wochenschr., 1895, No. 6.

[5] See Naunyn, Mittheilungen aus der medicin. Klinik zu Königsberg, Leipsic, 1888.

*mold organisms* are present. This last corresponds to the findings of A. Rose and myself.

**Yeast.**—A few isolated yeast-cells are found in the normal stomach. In ectasia or atony of marked degree the yeast-cells are numerous, arranged in colonies, and are in active process of germination (Fig. 95).

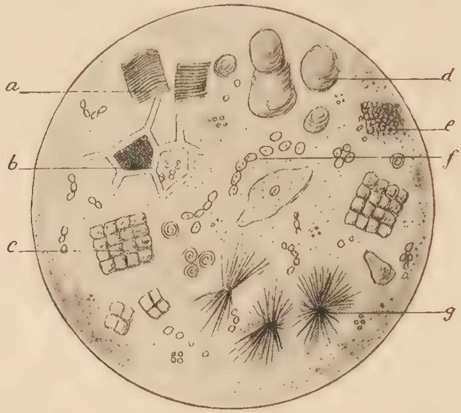

Fig. 95.—Benign ectasia. Yeast-cells and sarcinæ are prominent: *a*, Muscle-fiber; *b*, plant-cells; *c*, sarcinæ; *d*, starch granules; *e*, degenerated sarcinæ; *f*, yeast-cells; *g*, fat crystals.

**Sarcinæ.**—These occur usually in cubes or bales, and are only pathologic if present in large numbers, as in benign ectasia or atony (Fig. 95) in the presence of hydrochloric acid.

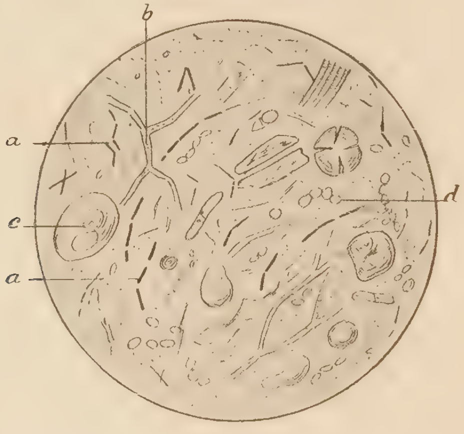

Fig. 96.—*a*, Boas-Oppler bacilli; *b*, leptothrix; *c*, potato-cell; *d*, yeast-cells.

They are *rare in ectasia from cancer*, and, if present, occur usually in cancer developed on an ulcer. Their presence in large numbers is an aid to diagnosis.

**Boas-Oppler Bacilli.**—They are unusually long, non-motile bacteria, and are characterized by their large size and end-to-end arrangement (Fig. 96).

They must be differentiated from the Leptothrix buccalis by Gram's solution, with which they stain brown, the leptothrix, blue. Kaufmann's[1] investigations prove the Boas-Oppler bacillus has the power of generating lactic acid from different sugars. Stagnation with lactic acid fermentation is *not specific* of pyloric carcinoma, but depends on the absence of hydrochloric acid and the presence of stagnation. These conditions exist in stenotic gastritis.

These bacilli have been found occasionally in stomach-contents that contain free hydrochloric acid (Rosenheim). Kaufmann has demonstrated them in 19 out of 20 cases of carcinoma.

The presence of these bacilli, with pronounced lactic acid fermentation and taken in connection with clinical symptoms, is very significant.

**Epithelial Cells, Particles of Tumor, and Fragments of Gastric Mucosa.**—Single cells cannot be diagnosed as cancer-cells, but cell-nests must be found. These are rarely discoverable.

Occasionally, after aspiration of the fasting stomach or test-meal, or after lavage, or in the vomitus, small particles of tissue may be found, which on staining may reveal the nature of a tumor.

Hemmeter recommends in suspected cases, first, thorough lavage and rectal feeding for a day; then passing the tube, moving it about actively, and subsequent aspiration of the fasting stomach. This is to be followed by lavage. All tissue fragments are to be examined.

Einhorn finds, especially after lavage, or at times after aspiration, small pieces of mucous membrane which he stains and examines. Some point to erosions, others to other affections. The fragment may be normal mucosa, or there may be proliferation of the connective tissue, or of the glands, or atrophy (partial or complete), or vacuolization. These conditions are illustrated in their appropriate chapters.

A positive judgment cannot be given from this examination, as only a small area may be actually involved and no changes observed in the gastric secretion. On the other hand, a bit of normal mucosa may be aspirated from a diseased organ.

**Mold.**—Mold in the stomach has been little referred to in literature as a pathologic condition except by Talma, A. Rose, Naunyn, Einhorn,[2] and Knapp.[3] In an article on "Dilatation of the Stomach"[4] I have already referred to it.

Einhorn has found it in the wash-water of the empty stomach as blackish-gray or brownish-green flakes of varying number; while Knapp describes it as coloring the chyme a yellowish green or dark red, and states that it has been mistaken respectively for bile or blood from its macroscopic appearance. He emphasizes the necessity for appropriate tests for bile and blood, and not the diagnosis from appearances. I can substantiate this in one case at least, in which the reddish-brown material proved to be mold.

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[1] Weiner klin. Wochenschr., 1895, No. 8.

[2] Medical Record, June 6, 1900.

[3] Organacidia Gastrica, Sept. 6, 1902.

[4] Medical News, Aug. 16, 1904.

The mold flocculi consist of clusters of spores and mycelia, sometimes mixed with mucus and epithelial cells. Crystals are also found, which Knapp considers to be segments of mold filaments.

The mold generally found has been identified by E. K. Dunham as the Penicillium glaucum, though Knapp reports in addition the Oïdium albicans and the aspergillus groups. These fungi probably adhere quite closely to the mucosa and may involve considerable areas (Fig. 97).

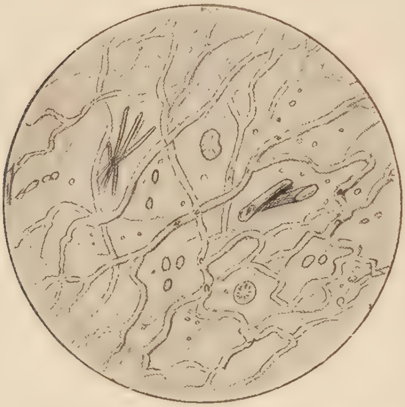

Fig. 97.—Green mold follicle, mycelia, spores, and crystals.

Einhorn reports mold formation in hyperchlorhydria, in some cases attended with hypersecretion, and also in gastralgia with normal or reduced gastric secretion.

Knapp holds that the presence of organic acids in the stomach has a decided bearing, and that succinic acid and mold go hand in hand, the presence of the former being conclusive. His test for succinic acid is as follows:

Extract 1 c.c. of filtered chyme with 4 c.c. of ether, and float this extract on a solution of ferric chlorid (1 drop of a 10 per cent. ferric chlorid to 2 c.c. of distilled water) in a narrow test-tube. At the line of junction is a dark mahogany-red ring. He further describes symptoms in many respects resembling severe hyperchlorhydria, with spasm of the pylorus, and believes the condition influenced by saccharine material in the chyme.

In the experience of A. Rose and the author, *diminished motility of the stomach* is a marked factor in favoring the growth of mold. I have found it in cases of atonic ectasia, producing many of the symptoms of chronic gastritis, and again in benign stenosis with hypersecretion. The subject requires still further investigation.

Rose finds the administration of 1 minim (0.059 c.c.) doses of beechwood creosote or carbonate of creosote 5 grains (0.3) t. i. d. of value, and lavage is, of course, indicated, preferably with warm water —1 liter followed by lavage with nitrate of silver 1 : 2000, or spraying the stomach with the latter, as suggested by Einhorn.

The general treatment should be according to the other conditions present.

## DETERMINATION OF THE ABSORPTIVE FUNCTION OF THE STOMACH

The absorptive function of the stomach is usually tested by the method of Penzoldt and Faber, as follows: 0.2 of potassium iodid is administered in a gelatin capsule, and the saliva or urine examined every minute or two with starch-paper and fuming nitric acid. The strip of paper is moistened with saliva or urine and then touched with a drop of the acid. A *violet or blue color is the reaction.* It takes six and one-half to fifteen minutes before the reaction appears in normal conditions. In pathologic cases it is retarded.

The test should be made on an empty stomach, as with the full organ it is retarded. It is self-evident that the gastric digestion of proteins—and hence their absorption—is delayed in cases of subacidity or anacidity, while these conditions are not so important for the digestion of carbohydrates.

In some cases the *absorption of iodid is normal,* though we *know protein digestion* is interfered with, so that I do not consider the test in every case reliable. Hershell gives a capsule containing 2 decigrams of powdered rhubarb. Under normal conditions the urine gives a red color with liquor potassæ. They are, however, the best tests so far known.

**Motor Functions of the Stomach.**—By this we mean the *peristaltic action of the stomach* which *expels its contents* into the intestine. The impairment of the motor power is *fully* as, and in many cases *more important* than, damage to the secretory functions.

The best method of testing the motor power is by the test-meal. Leube's is the oldest method. He administered a plate of soup, a beefsteak, and a roll. If the stomach was found empty seven hours later and nothing could be washed out, it indicated that its motor power is sufficiently active. If the food remains in the stomach longer, the motor power is reduced.

We must remember that in pyloric stenosis the motor power may be really increased in endeavoring to overcome the obstacle, but that food remains in the stomach for an abnormal length of time; strictly speaking, it is a relative motor insufficiency. If five hours after a test-meal a small amount of chyme is aspirated, the motor power is good. If large quantities are found six hours after the meal, the motor function is absolutely (or if stenosis, relatively) decreased. Greater degrees of insufficiency may be present. For example, lavage is performed and a test-supper administered, say, at 12 P. M. and the contents aspirated seven or eight hours later. In one case there may be a small quantity of food remaining, and in another case a large amount; while in another, none. I have found chopped spinach, a few raisins, or a piece of fig an addition to the test, as they are readily recognized. Boiled rice can be added.

Boas recommends cold meat with rolls and butter and a large cup of tea.

It may be more convenient to follow the same procedure, but give the meal at lunch-time. Some employ the *test-breakfast. Two hours later* the stomach *should be empty.* If 100 c.c. or more are found at the end of an hour, or varying quantities at the end of two hours, it shows different degrees of motor insufficiency. The test-meal is more accurate. I sometimes administer a test-supper and aspirate in the morning to test the motor function, following immediately with the test-breakfast to examine the secretory function.

*Salol Test* (*Ewald and Siever's Method*).—Salol is not decomposed in the stomach, but in the alkaline medium of the intestine. Here it is split up, and the salicylic acid is absorbed and eliminated in the urine as salicyluric acid. The latter is recognized by testing the urine with neutral ferric chlorid solution, which gives a violet color with this acid.

The patient takes salol 15 grains (1.0) in two gelatin capsules half an hour after a light meal. The bladder is first emptied. Thereafter he urinates every half-hour for about two hours, and the different specimens of urine are tested with the iron solution. Under normal conditions the reaction appears in from thirty to seventy-five minutes. In retarded motility it takes two hours or more.

Ewald treats the urine with ether and examines the ethereal residue; while Einhorn moistens a piece of filter-paper with the urine and touches the middle of it with the iron solution.

Huber suggests to determine the time required for the complete disappearance of the reaction in the urine. The longer the time required for the salol to be absorbed and entirely eliminated through the urine, the longer it has remained within the stomach. He found that normally the excretion of the salicyluric acid after salol was administered lasted twenty-four hours; in patients with diminished motor function it lasted forty-eight hours or more.

*Iodipin Test.*—Iodipin is decomposed in the intestine. Heichelheim gives 1.6 gm. iodipin in gelatin capsules at breakfast. The saliva is then examined every fifteen minutes for iodin by starch-paper and fuming nitric acid. In normal cases the reaction appears within an hour.

*Klemperer's Oil Test.*—Oil is not absorbed in the stomach. After washing the stomach, 100 c.c. of pure olive oil are poured into the empty organ. Two hours later the stomach is thoroughly aspirated. The difference between the original quantity of oil and that withdrawn indicates the condition of the motor function. Normally at this time only 20 to 40 c.c. of oil should be aspirated.

*Einhorn's Gastrograph.*—Einhorn has invented a deglutible ball, arranged with an electric circuit, so that the movements which mix and break up the food can be registered.

*Hemmeter-Moritz Method.*—This method has been independently employed by Hemmeter and Moritz.

A thin, deglutible bag is attached to an esophageal tube. The bag is then blown up and connected with a tambour on the Ludwig kymograph, which registers all the movements with a pen. A pneumograph is tied about the chest to record the respiratory movements as a basis of comparison. The muscular contractions of the stomach are demonstrated on the record as independent of the respiration.

These instruments are of interest scientifically, but their practical value has not been demonstrated.

# CHAPTER VII

# DIET

THE study of nutrition, both in health and disease, is important, but it will only be possible to enunciate the general principles.

There are three groups of food stuffs—Proteins, carbohydrates, and fats—procured from the animal and vegetable kingdoms, which in conbination furnish the most suitable form of nourishment. Climatic conditions and environment have an influence on the requirements for nutrition. In extremely cold climates the Eskimos have lived for many generations on nearly an exclusively nitrogenous diet, in which fat, which produces the greatest number of heat units, predominates. In hot regions many of the races live principally on a non-nitrogenous diet. We also know that vegetarians live and thrive on carbohydrates.

A mixed diet is the most suitable form of nourishment.

## DIET IN HEALTH

Voit has emphasized the fact that the smallest amount of protein, with non-nitrogenous food added, that will keep the body in continual vigor is the ideal diet. He holds that a healthy adult of average weight should ingest 100 gm. of albumin, 50 gm. of fat, and 450 gm. of carbohydrate in twenty-four hours; others place the requirement for protein as considerably higher. A small proportion of the food serves the purpose of reconstructing the tissue waste, while the major part is used for generating the heat required for the maintenance of life. It is, therefore, customary to speak of the necessary amount of heat units during the twenty-four hours instead of the quantity of food.

A calorie (or heat unit) may be defined as the amount of heat required to raise 1 gram of water 1° C. This is a small calorie. A large calorie is the amount necessary to raise 1 kilogram of water 1° C. Hence a large calorie equals 1000 small calories.

| | | | | |
|---|---|---|---|---|
| 1 gm. carbohydrate | yields | 4.1 | large | calories. |
| 1 gm. fat | " | 9.3 | " | " |
| 1 gm. protein | " | 4.1 | " | " |

In order to calculate the calorie value of any kind of food, the number of grams of albumin that are contained in it are multiplied by 4.1; the grams of carbohydrate by 4.1; the grams of fat by 9.3. These are added together and give the total calorie value of the food. For example:

| | | | | |
|---|---|---|---|---|
| 100 gm. | albumin | × 4.1 = | 410 | calories. |
| 50 " | fat | × 9.3 = | 465 | " |
| 450 " | carbohydrate | × 4.1 = | 1845 | " |
| | | | 2720 | total calories. |

The calorie value of vegetable protein is slightly less than that of animal protein; 50 gm. of fat about equal 113 gm. of starch in calorie value.

Riegel holds that a human being at rest demands about 35 calories per kilogram of his body weight, and a person performing light work about 40 calories per kilo. From this estimate, the calorie value of the food of an individual weighing 50 kilos is from 1750 to 2000 calories. The weight of the patient must, therefore, be known in order to select the correct amount of nourishment.

Rubner states that different articles of food can replace each other according to their calorie value, and that it is immaterial in what form the calories are introduced into the organism. This may be taken advantage of temporarily in certain pathologic conditions when it is necessary to limit some special variety of food, such as the carbohydrates. On the other hand, a certain amount of protein is necessary for the organism, while *unquestionable damage* can be done *by excess* in this direction.

The results of scientific study are opposed to the prevailing dietary standards, especially in regard to protein foods. It is true that no other form of food can take the place of proteins, for a certain quantity is needed each day to replace the loss of tissue material broken down, and our choice of the varied articles of diet should be regulated by the amount of protein they contain. It is not necessary, however, that they should exceed the other foods in amount or approach them in quantity.

Russell H. Chittenden [1] has clearly demonstrated by his scientific researches that the recommended dietary standards are excessive in quantity,[2] especially in regard to proteins. They do not undergo complete oxidation in the body like non-nitrogenous foods, but there is left behind a residue of non-combustible matter, crystalline nitrogenous products, which ultimately, if occurring in excess of the requirements of the body, prove injurious to the gastro-intestinal tract, liver, kidneys, and the circulatory and nervous system.

The fats and carbohydrates are easily eliminated, becoming carbonic acid gas and water. Overfeeding with a tendency to obesity is an evil, as the fat acts as a mechanic obstacle to the activity of the body and interferes with the movements of the heart and other organs; and, in addition, fatty degeneration may occur. *Potential energy*, however, can be fully as advantageously met by the non-nitrogenous foods, carbohydrates, and fats.

Though the author has been criticized for his approval of Chittenden's low protein diet in health, he must confess that, from personal experience and prolonged investigation, he must continue to advocate Chittenden's methods. Unquestionably a considerable percentage of temporary disturbances or permanent damage in the gastro-intestinal tract, the liver, kidneys, and in the arterial and nervous systems can be imputed to excessive protein diet, from the intestinal putrefaction resulting therefrom. Rheumatic affections, gravel, and rheumatoid arthritis, the writer believes, are influenced by the same factor.

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[1] Nutrition of Man.

[2] Physiologic Economy in Nutrition.

Fletcher has demonstrated on himself that deliberation in eating necessitated by the habit of thorough insalivation, results in the occurrence of satiety on the ingestion of a comparatively small amount of food, and hence excess is avoided.

It is clear that a man of 170 lbs. weight has more protein tissue to nourish than one of 130 lbs., and consequently what will suffice for the latter will not for the former. Dietary standards are merely approximate and depend on the physical work to be performed, the body weight, sex, age, climate, etc. There is doubtless a specific coefficient of nutrition characteristic of the individual.

Chittenden demonstrated on professional men, soldiers, and athletes that they could perform their duties with greater vigor, and their strength as measured by tests was increased under diminished protein diet; in fact, with about one-half the amount as compared with the standards suggested.

### CHITTENDEN'S TABLE

| 60 gm. of protein are contained in— | Fuel value.[1] Calories. |
|---|---|
| $\frac{1}{2}$ lb. fresh lean beef (loin) | 308 |
| 9 hens' eggs | 720 |
| $\frac{1}{5}$ lb. sweetbread | 660 |
| $\frac{3}{4}$ lb. fresh liver | 432 |
| $\frac{7}{8}$ lb. lean smoked bacon | 1820 |
| $\frac{3}{4}$ lb. halibut steak | 423 |
| $\frac{1}{2}$ lb. salt codfish (boneless) | 245 |
| 2$\frac{1}{5}$ lbs. oysters, solid | 506 |
| $\frac{1}{2}$ lb. American pale cheese | 1027 |
| 4 lbs. (2 quarts) of whole milk | 1300 |
| $\frac{5}{6}$ lb. uncooked oatmeal | 1550 |
| 1$\frac{1}{4}$ lbs. shredded wheat | 2125 |
| 1 lb. uncooked macaroni | 1665 |
| 1$\frac{1}{3}$ lbs. white wheat bread | 1520 |
| 1$\frac{1}{4}$ lbs. crackers | 2381 |
| 1$\frac{2}{3}$ lbs. flaked rice | 2807 |
| $\frac{3}{5}$ lb. dried beans | 963 |
| 1$\frac{7}{8}$ lbs. baked beans | 1125 |
| $\frac{1}{2}$ lb. dried peas | 827 |
| 1$\frac{1}{12}$ lbs. potato-chips | 5728 |
| $\frac{2}{3}$ lb. almonds | 2020 |
| $\frac{2}{5}$ lb. pine-nuts, pignolias | 1138 |
| 1$\frac{2}{3}$ lbs. peanuts | 3584 |
| 10 lbs. bananas (edible portion) | 4600 |
| 10 lbs. grapes | 4500 |
| 11 lbs. lettuce | 990 |
| 15 lbs. prunes | 5550 |
| 33 lbs. apples | 9570 |

The standards of 100 gm. of protein or more mean the *excretion of excessive nitrogen* through the urine. Chittenden[2] found by experiment that the average need of protein food by adults is fully met by a daily metabolism equal to an exchange of 0.12 gm. of nitrogen per kilogram of body weight. This means a catabolism of ¾ gm. of protein matter daily per kilogram. The intake of protein food must be somewhat in excess of protein catabolism, since not all of the protein is available, and this is a variable amount, depending on the proportion of animal and vegetable foods with their different degrees of digestibility and availability. The required intake of protein Chittenden places at 0.85 gm. per kilogram of body weight, giving a maximum for safety. Hence, for a man weighing 70 kilos (154 lbs.) there would be required daily 59.5 gm. (say, 60 gm.) of protein food to meet the needs of the body. This is about one-half the Voit standard, and far below that of many other so-called diets. As the specialist is so often consulted as to the proper diet to maintain the health of the body, I quote Chittenden at some length, being a firm believer in his principles.

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[1] Fuel value of the quantity needed to furnish 60 gm. of protein.

[2] Physiologic Economy in Nutrition; Nutrition of Man.

The daily protein requirement of 60 gm. can be obtained from ½ pound of uncooked lean meat, of which loin steak is the type. Lamb, veal, poultry, or lean flesh of any variety, of equivalent weight, will approximately furnish the same amount of protein.

Fish, such as halibut steak, and liver require ¾ pound, and of sweetbreads ⅘ pound is necessary.

Of salt codfish ½ pound is equivalent to the same weight of fresh beef, while of lean smoked bacon ⅞ pound is necessary.

*Three hen's eggs* furnish one-third the amount of protein required in twenty-four hours. Dried peas and beans, almonds, and pine-nuts are as rich in proteins as the above-mentioned animal foods, and essentially the same weight is called for to provide the daily requirement of protein. The same is true of cheese, the composition of ½ pound being equivalent to the same amount of protein, but of much *higher caloric value* than the equivalent weight of fresh beef.

There are some differences in digestibility which tend to lower slightly the availability of the vegetable products, also of the cheese, which necessitates a slight increase in the amount of these foods to equal the protein value of the equivalent weight of lean beef.

Certain foods are poor in proteins, such as fruits, bananas, grapes, prunes, apples, etc., lettuce, and, to a less degree, potatoes. These are all palatable, but add *little to the proteins*, even when given in large amount.

There is a radical difference between the animal foods and those of vegetable origin, in that the fuel value necessary to furnish the 60 gm. of protein is small in the former, as compared with that of the vegetables; ½ pound of lean beef, with its 60 gm. of protein, has, for example, a fuel value of only 308 calories, while ⅔ pound of almonds has one of 2020 calories; ½ pound of cheese has one of 1027 calories; ½ pound of dried peas, 827 calories. This is due to the proportion of fat or oil present. With fat meat, such as bacon, the calorie value rises in proportion to the increase of fat, the protein decreasing to a greater or less degree.

A high protein (animal) diet cannot serve for man. In a male, for example, with a weight of 70 kilos, and requiring 2800 calories, it would necessitate the ingestion of 4½ pounds of beef to secure this result, or nine times more protein than is necessary for the system.

Certain vegetable foods on the diet-list, such as flaked rice, crackers, and shredded wheat, contain proteins, with carbohydrates and fat in such proportion that the energy requirement would be met with essentially by the same quantity as served to furnish the necessary protein. In potatoes and bananas the fuel value predominates over the protein. The ideal diet is an admixture, such as wheat bread with butter or fat bacon to add to its calorie value, shredded wheat with cream, crackers with cheese, bread and milk, eggs with bacon, meat with potatoes, etc.

Two quarts of milk will furnish half the requirement of an average man, and reinforced by a 1-pound loaf of wheat bread, gives the requisite amount. A better combination is ¼ pound of lean beef, ⅔ pound of bread, and ½ pound of butter.

According to Chittenden, for a man of average weight of 70 kilos (154 pounds), to provide the requisite quantity of food—*i. e.*, 60 gm. of protein and 2800 calories—the following is a sample dietary:

| *Breakfast:* | Protein, grams. | Calories. |
|---|---|---|
| 1 shredded wheat biscuit (30 gm.) | 3.15 | 106 |
| 1 teacup of cream (120 gm.) | 3.12 | 206 |
| 1 German water roll (57 gm.) | 5.07 | 165 |
| 2 1-inch cubes of butter (38 gm.) | 0.38 | 284 |
| ¾ cup of coffee (100 gm.) | 0.26 | |
| with | | |
| ¼ teacup of cream (30 gm.) | 0.78 | 51 |
| 1 lump of sugar (10 gm.) | ... | 38 |
| | 12.76 | 850 |

| *Lunch:* | Protein, grams. | Calories. |
|---|---|---|
| 1 teacup home-made chicken soup (144 gm.) | 5.25 | 60 |
| 1 Parker House roll (38 gm.) | 3.38 | 110 |
| 2 1-inch cubes of butter (38 gm.) | 0.38 | 284 |
| 1 slice lean bacon (10 gm.) | 2.14 | 65 |
| 1 small baked potato (2 ounces—60 gm.) | 1.53 | 55 |
| 1 rice croquette (90 gm.) | 3.42 | 150 |
| 2 ounces maple syrup (60 gm.) | ... | 166 |
| 1 cup tea with 1 slice lemon | ... | ... |
| 1 lump sugar (10 gm.) | ... | 38 |
| | 16.10 | 928 |

| *Dinner:* | Protein, grams. | Calories. |
|---|---|---|
| 1 teacup cream of corn soup (130 gm.) | 3.25 | 72 |
| 1 Parker House roll (38 gm.) | 3.38 | 110 |
| 1 1-inch cube of butter (19 gm.) | 0.19 | 142 |
| 1 small lamb chop broiled, lean meat (30 gm.) | 8.51 | 92 |
| 1 teacup of mashed potato (167 gm.) | 3.34 | 175 |
| Apple-celery lettuce salad with mayonnaise dressing (50 gm.) | 0.62 | 75 |
| 1 Boston cracker split, 2 in. in diameter (12 gm.) | 1.32 | 47 |
| ½-inch cube American cheese (12 gm.) | 3.35 | 50 |
| ½ teacup of bread pudding (85 gm.) | 5.25 | 150 |
| 1 demi-tasse coffee | ... | ... |
| 1 lump sugar (10 gm.) | ... | 38 |
| | 29.21 | 951 |

The total with the dietary for the day amount to 58.07 gm. of protein and 2729 calories.

These figures are to be considered only approximately correct.

If a little more protein is required without changing materially the fuel value, a boiled egg can be added to the breakfast. An average egg of 53 gm. contains 6.9 gm. of protein and increases the fuel value by 80 calories. If more vegetable protein is desired, a soup of split-peas can be introduced without changing to any great degree the calories; thus, one teacup of split-pea soup (1.44 gm.) contains 8.64 gm. of protein, while the fuel value is only 94 calories.

The addition of 1 banana (160 gm.) will increase fuel value 153 calories, but will only add 2.28 gm. of protein.

If the fuel value is to be increased without change in the protein contents of the food, recourse can be had to butter, fat of meat, additional oil in salads, or to syrup and sugar.

Wheat products abounding in starch still show a large proportion of protein; thus, shredded wheat biscuit (1 ounce), which is a type of many wheat preparations from bread and biscuit to various breakfast foods, yield about 3 gm. of protein per ounce and 100 calories; 1 ounce of olive oil contains 100 calories. Potato, chiefly a carbohydrate, yields nitrogen the equivalent of about ¾ gm. of protein per ounce. If a large volume is desired without much increase in real food value, there are green foods, such as lettuce, celery, greens of various sorts, fruits, such as apples, grapes, oranges, etc.

Meat augments largely the intake of protein and adds relatively a small amount to the fuel value.

In edible nuts the content of protein is high, in some cases higher than in fresh beef; while carbohydrates and fat are large in amount, as in almonds and peanuts.

In *pine-nuts and Brazil nuts carbohydrates* are small in quantity as compared with peanuts, almonds, and walnuts, an important fact where a vegetable rich in protein is desired, but with freedom from starch (see table).

| | Proteid present. | Carbohydrate present. | Fat present. | Water present. | Mineral present. | Fuel value per lb. |
|---|---|---|---|---|---|---|
| Almonds (edible portion).... | 21.0 | 17.3 | 54.9 | 4.8 | 2.0 | 3030 |
| Peanuts (edible portion)..... | 25.8 | 24.4 | 38.6 | 9.2 | 2.0 | 2560 |
| Pine-nuts (edible portion)... | 33.9 | 6.9 | 49.4 | 6.4 | 3.4 | 2845 |
| Brazil nuts (edible portion).. | 17.0 | 7.0 | 66.8 | 5.3 | 3.9 | 3265 |
| Soft-shell walnuts........... | 16.6 | 16.1 | 63.4 | 2.5 | 1.4 | 3285 |

United States Department Agriculture Bulletin No. 28.

Green vegetables, such as spinach, help the bowel action.

Natural sugars are of value, especially such as occur in oranges, grapes, prunes, dates, plums, and bananas, and to a less degree in apricots, peaches, pears, apples, figs, strawberries, raspberries, and blueberries.

Chittenden[1] has given considerable study as to the value of the pineapple. He has demonstrated that the juice, in fresh condition, contains an enzyme or enzymes of a proteolytic nature, active in either an alkaline carbonate or in an acid medium. It was found to be most active at 122° to 140° F., and 158° F. destroys it, but it possesses digestive power at 86° F. Leucin, tyrosin, proteoses, and peptones were formed, so that its enzyme is more closely related to trypsin than to pepsin. It is capable of digesting a large amount of protein with great rapidity, and hence may be valuable in conditions with deficient hydrochloric secretion.

Apples, when ripe and well masticated, are good, and a baked apple is wholesome.

There is occasionally an idiosyncrasy to strawberries, and some suffer from fermentation from various fruits.

**Soy Gruel Flour.**—The *Soy bean*, which is largely employed by the Chinese and Japanese as an article of food, contains about 25 per cent. of protein. J. Ruhräh[2] has recently secured the manufacture of a *flour* made from this bean, which he advocates as a useful addition to the diet of infants when it is desired to supply an early digestible protein free from starch. Friedenwald has also recommended it as a source of protein for diabetics who tire of meats, and it has proved useful in the form of a weak gruel in diarrheas and intestinal disturbances, and when meat is contraindicated. On account of the removal of the coarse fibrous hulls, the per cent. of protein in the flour is nearly one-third greater than in the whole bean. The analysis of the soy gruel flour is as follows:

| | Per Cent. |
|---|---|
| Protein (N. x 6.25) | 44.64 |
| Fat | 19.43 |
| Mineral matter | 4.20 |
| Moisture | 5.26 |
| Crude fiber | 2.35 |
| Cane-sugar | 9.34 |
| Non-nitrogenous extract | 14.78 |
| Starch | None |
| Reducing sugars | None |
| Polarization normal weight due to optically active substance other than cane-sugar (included in proteins and non-nitrogenous extract) | 7.68° |

Each ounce of the flour yields about 13 *grams of protein* and 120 *calories* and it can be used as a gruel, in broths, and in making biscuits or muffins. It may be mixed with cereals, barley jelly, cream of wheat, etc. The following table shows the composition and calorie value of the gruels made from this flour:

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[1] Journal of Physiology, xv, p. 249.

[2] Archiv. Pediat., July, 1909; Jour. Amer. Med. Assoc., July 10, 1909, and May 21, 1910; Soy Bean Cookery, Med. Rec., Sept. 23, 1911; Amer. Jour. Med. Sci., Dec. 1910.

| Amount. | Protein per cent. | Fat per cent. | Sugar per cent. | Calories. |
|---|---|---|---|---|
| ¼ oz. (1 level tablespoonful to quart)........ | 0.35 | 0.15 | 0.08 | 30 |
| ½ oz. (2 level tablespoonfuls to quart)....... | 0.70 | 0.30 | 0.15 | 60 |
| ¾ oz. (3 level tablespoonfuls to quart)....... | 1.00 | 0.45 | 0.23 | 90 |
| 1 oz. (4 level tablespoonfuls to quart)....... | 1.40 | 0.60 | 0.30 | 120 |
| 2 oz. to quart.......................... | 2.80 | 1.20 | 0.60 | 240 |
| 3 oz. to quart.......................... | 4.20 | 1.80 | 0.90 | 360 |
| 4 oz. to quart.......................... | 5.60 | 2.40 | 1.20 | 480 |
| 5 oz. to quart.......................... | 7.00 | 3.00 | 1.50 | 600 |
| 6 oz. to quart.......................... | 8.40 | 3.60 | 1.80 | 720 |
| 7 oz. to quart.......................... | 9.80 | 4.20 | 2.10 | 840 |
| 8 oz. to quart.......................... | 11.00 | 4.80 | 2.40 | 960 |

The calorie value of the gruel can be increased by adding condensed milk, sugar, or cereals.

(1 oz.) 4 level tablespoonfuls of barley or wheat flour will add 100 calories.
(1 oz.) 4 level tablespoonfuls of oat flour will add 115 callories.
(1 oz.) 2 level tablespoonfuls of granulated sugar will add 100 calories.
(1 oz.) 3 level tablespoonfuls of milk-sugar will add 100 calories.
Two ounces of sugar in a 32-oz. mixture = 6 per cent.

The following cooking directions are advised by Ruhrah:

*Gruels.*—A quart of gruel is made by boiling from 1 level tablespoonful to 6 ounces of the soy gruel flour in 1 quart of water for fifteen minutes, adding water to make up for loss by evaporation. Salt should be added to taste.

| Composition. | Protein per cent. | Fat per cent. | Carbohydrates per cent. |
|---|---|---|---|
| One level tablespoonful to quart..................... | 0.35 | 0.15 | 0.08 |
| One oz. (4 tablespoonfuls to quart).................. | 1.40 | 0.60 | 0.30 |

These gruels do not thicken during cooking, as they contain no starch, and readily settle on standing. This may be overcome by adding 1 to 2 heaping teaspoonfuls of barley, oat, or wheat gruel flour before cooking, which will add from 0.6 to 1.2 per cent. starch to the gruels, and also slightly increase the percentage of protein.

*Broths.*—Add from 1 to 8 ounces of the flour to 1 quart of beef, mutton, veal, or chicken broth and boil for fifteen minutes, adding water to make up for loss by evaporation; or, boil the same quantity of the soy flour for one-half hour with 1 quart of water, to which has been added a piece of ham, bacon, or salt pork to give flavor. Each ounce of the flour will add to the broth about 13 grams of protein, and 120 calories, or, in percentages, add 1.4 per cent. protein, 0.6 per cent. fat, and 0.3 per cent. carbohydrates. A broth made with 6 ounces of the soy flour to the quart would be half as rich in protein and fat as steak.

*Muffins.*—To make muffins from the soy flour, take 1¼ teacupfuls of the soy flour, ¼ teacup of wheat flour, ½ teaspoonful of salt, 2 eggs, 1 teacupful of sweet milk, 2 rounded teaspoonfuls of baking-powder, and 1½ tablespoonfuls of melted, but not hot, butter. Beat well together, adding the melted butter last, and bake in gem pans in a hot oven. This will make about 12 muffins, which will contain about 150 grams of protein, and which will yield about 1800 calories, of which the carbohydrates produce but 280.

The soy flour, being nitrogenous, requires the addition of some wheat flour in making muffins, as above. The mixture of wheat and soy flours in this formula will contain about 36 per cent. protein and 20 per cent. carbohydrates, against 14 per cent. protein and from 60 to 70 per cent. carbohydrates in gluten flour. The proportion of protein to carbohydrates is from 8 to 10 times as large in the mixed soy and wheat flour as in gluten flour.

The author believes that in this soy gruel we have a valuable addition to our dietary, and believes it useful in typhoid fever and in many other conditions.

*A diet conforming to true nutritive requirements must tend toward vegetable food if excess in proteins is to be avoided.*

There is less need for food in hot weather, especially for fat, when lighter foods and less calories are required.

We must also remember that the excessive use of salt strains the kidneys.

The following table, modified from that of Koenig and others, gives the chemic composition of different foods and the heat units which they produce.

### CHEMIC COMPOSITION OF COMMON FOOD SUBSTANCES

I. *Meats and Game.*

| | Per cent. Nitrogenous (proteid). | Per cent. Fat. | Per cent. carbohydrate (nitrogen free). | Calories per 100. |
|---|---|---|---|---|
| Beef (very fat) | 17.19 | 26.38 | .... | 315.81 |
| Beef (lean) | 20.78 | 1.50 | .... | 99.15 |
| Veal (fat) | 18.88 | 7.41 | 0.07 | 146.61 |
| Veal (lean) | 19.84 | 0.82 | .... | 86.97 |
| Mutton (fat) | 14.80 | 36.39 | 0.05 | 399.31 |
| Mutton (lean) | 17.11 | 5.77 | .... | 123.81 |
| Pork (fat) | 14.54 | 37.34 | .... | 406.88 |
| Pork (lean) | 20.25 | 6.81 | .... | 146.36 |
| Westphalia ham | 23.97 | 36.48 | 1.50 | 453.69 |
| Salted ham | 22.32 | 8.68 | .... | 173.23 |
| Smoked beef | 27.10 | 15.35 | .... | 253.76 |
| Smoked beef tongue | 24.31 | 31.61 | .... | 393.64 |
| Pulverized meat | 64.5 | 5.24 | 2.28 | 322.53 |
| Sweetbread | 22.0 | 0.4 | .... | 93.92 |
| Chicken (fat) | 18.49 | 9.34 | 1.20 | 167.58 |
| Chicken (lean) | 19.72 | 1.42 | 1.27 | 99.10 |
| Capon | 23.32 | 3.15 | 2.49 | 135.11 |
| Duck (wild) | 22.65 | 3.11 | 2.33 | 131.36 |
| Partridge | 25.26 | 1.43 | .... | 116.85 |
| Pigeon | 22.14 | 1.00 | 0.76 | 100.02 |
| Hare | 23.34 | 1.13 | 0.19 | 107.08 |
| Venison | 19.77 | 1.92 | 1.42 | 105.44 |

II. *Fish.*

| | Per cent. nitrogenous (proteid). | Per cent. fat. | Per cent. carbohydrate (nitrogen free). | Calories per 100. |
|---|---|---|---|---|
| Eel | 12.83 | 28.37 | 0.53 | 312.93 |
| Pike | 18.34 | 0.51 | 0.63 | 83.57 |
| Carp | 20.61 | 1.09 | .... | 94.64 |
| Shellfish | 17.09 | 9.34 | .... | 156.93 |
| Halibut | 11.94 | 0.25 | 0.45 | 53.66 |
| Salmon | 15.01 | 6.42 | 2.85 | 132.93 |
| Sardellen | 22.30 | 2.21 | 0.45 | 113.83 |
| Oysters | 4.95 | 0.37 | 2.62 | 34.39 |
| Fresh herring | 10.11 | 7.11 | .... | 106.15 |
| Salt herring | 18.90 | 16.89 | 1.57 | 247.61 |
| Caviar | 31.36 | 15.61 | 2.23 | 279.76 |

### III. *Dairy Products*

| | Per cent. nitrogenous (proteid). | Per cent. fat. | Per cent. carbohydrate (nitrogen free). | Calories per 100. |
|---|---|---|---|---|
| Cows' milk........ | 3.41 to 4.3 | 3.0 to 3.8 | 3.7 to 4.81 | 56.41 to 71.93 |
| Cream............ | 3.61 | 26.75 | 3.52 | 276.01 |
| Buttermilk....... | 3.0 to 4.0 | .93 to 1.3 | 3.0 to 4.0 | 33.08 to 43.63 |
| Whey............ | .85 | .23 | 3.03 | 18 |
| Kumyss (cows' milk)........ | 3.65 | 2.07 | Lactic acid, .7<br>Alcohol, 1.9<br>Carbonic acid, 8. | 32.99 |
| Butter........... | 0.5 | 90 | 0.5 | 823.1 |
| Cheese (cream).... | 16.28 | 41.22 | 1.90 | 449.54 |
| Cheese........... | 34.99 | 11.37 | 5.40 | 269.06 |
| Eggs (hens')...... | 12.5 | 12.1 | 0.5 | 165 |
| White of egg...... | 12.67 | 0.25 | .......... | 54.22 |
| Yolk of egg....... | 16.24 | 31.75 | 0.12 | 355.99 |

### IV. *Cereals and Vegetables*

| | Per cent. nitrogenous (proteid). | Per cent. fat. | Per cent. carbohydrate (nitrogen free). | Calories per 100. |
|---|---|---|---|---|
| Wheat bread...... | 6.0 | 0.75 | 52.0 | 245 |
| Rye bread........ | 6.11 | 0.43 | 46.0 | 217.56 |
| Sago............. | 0.5 | traces | 86.5 | 356.70 |
| Wheat flour....... | 8.5 | 1.25 | 73.0 | 345.78 |
| Rye flour......... | 10.0 | 2.0 | 69.0 | 342.50 |
| Cakes............ | 11.0 | 4.60 | 73.30 | 387.09 |
| Roll............. | 6.82 | 0.77 | 43.72 | 213.87 |
| Zwieback......... | 9.5 to 13.0 | 1.0 to 3.0 | 75.0 | 356 (average) |
| Cauliflower....... | 2.0 to 5.0 | 0.4 | 4.0 | 35 |
| Potatoes.......... | 1.5 | .... | 20.0 | 88 |
| Asparagus........ | 2.0 | 0.3 | 2.5 | 20 |
| Carrots........... | 1.04 | 0.21 | 6.74 | 33.85 |
| Rice............. | 5.5 | 1.5 | 76.0 | 348.10 |
| Beans............ | 19.5 | 2.0 | 52.0 | 311.75 |
| Peas............. | 19.5 | 2.0 | 54.0 | 319.95 |
| Spinach.......... | 2.49 | .58 | 4.44 | 33.67 |
| Oatmeal.......... | 12.05 | 5.26 | 66.77 | 338.80 |
| Barley meal....... | 8.31 | 0.81 | 75.19 | 323 |
| Brussels sprouts... | 4.83 | 0.41 | 6.22 | 49.05 |
| Cabbage (white).. | 1.89 | 0.20 | 4.87 | 29.52 |
| Pickles.......... | 1.02 | 0.09 | 0.95 | 8.81 |

V. *Soups and Beverages*

| | Per cent. nitrogenous proteid. | Per cent. fat. | Per cent. non-nitrogenous carbohydrate. | Calories per 100. |
|---|---|---|---|---|
| Meat broth | 0.4 | 0.6 | .... | 7.10 |
| Meat juice (expressed) | 6.0 to 7.0 | 0.5 | 0.5 | 31.20 (average) |
| Beef-tea | 0.5 | 0.5 | | 6.6 |
| Leube's meat solution | 9 to 11 albumin and 1.7 to 6.5 pepton. | .... | .... | 86.5 (average) |
| Malt extract | 8.0 to 10.0 | .... | 55.0 | 258.30 |
| Milk soup with wheat flour | 5.0 | 3.25 | 15.0 | 112. |
| Barley soup | 1.5 | 1.0 | 11.0 | 60.96 |
| Rice pap with milk | 8.8 | 3.5 | 28.6 | 182.61 |
| Coffee | 3.12 | 5.18 | .... | 59.92 |
| Tea | 12.38 | .... | .... | 50.75 |
| Beer | 0.5 | 5.25 | 0.3 | 51.0 |
| Porter | 0.7 | 6.0 | 0.3 | 60. |

VI. *Fruits, Nuts, and Sugar*

| | Per cent. free acid. | Per cent. nitrogenous proteid. | Per cent. fat. | Chiefly sugar. | |
|---|---|---|---|---|---|
| | | | | Per cent. non-nitrogenous carbohydrate. | Calories per 100. |
| Apples | 0.82 | .... | .... | 7.22 | 29.6 |
| Pears | 0.20 | .... | .... | 8.24 | 33.78 |
| Plums | 1.50 | .... | .... | 4.68 | 19.18 |
| Peaches | 0.92 | .... | .... | 7.17 | 29.39 |
| Apricots | 1.16 | .... | .... | 4.69 | 19.22 |
| Grapes | 0.79 | .... | .... | 14.36 | 58.87 |
| Strawberries | 0.93 | .... | 0.45 | 6.78 | 31.88 |
| Chestnuts | .... | 5.48 | 1.37 | 38.34 | 192.11 |
| Cane-sugar | .... | 0.35 | .... | 93.33 | 382.65 |
| Beet-sugar | .... | .... | .... | 99.75 | 408.97 |
| Honey | .... | 1.20 | .... | 73.22 | 305.22 |

For the determination of the calorie value of each kind of food, the number of grams of albumin must be multiplied by 4.1, the grams of carbohydrate by 4.1, and the grams of fat by 9.3, and the multiples added will give the total calories as already described.

The following diet-lists of von Noorden[1] demonstrate the method of calculating calorie values, and will be found useful to fulfil their indications:

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[1] Berl. Klinik, 1838, J. 55.

I. *A Chiefly Milk Diet with Addition of Carbohydrates in Liquid Form*

| | Albumin (per cent.). | Fat (per cent.). | Carbohydrate (per cent.). | Calories per 100. |
|---|---|---|---|---|
| Milk, 1700 cc. | 70.2 | 66.3 | 69.7 | 1295 |
| Soup of tapioca flour, 30 gm. and 10 gm. albumose[1] | 10 | .... | 30 | 164 |
| Soup of 40 gm. wheat flour, with some of the milk, 10 gm. sugar, and one egg | 7.0 | 5.5 | 40 | 244 |
| Total | 87.2 | 71.8 | 139.7 | 1703 |

II. *A Chiefly Milk Diet with the Addition of Carbohydrates and Fat in Mushes and Soups*

| | Albumin (per cent.). | Fat (per cent.). | Carbohydrates (per cent.). | Calories per 100. |
|---|---|---|---|---|
| Good milk, 1500 cc. | 62 | 58.5 | 63 | 1056 |
| Soup of 15 gm. sago, 10 gm. butter, 1 egg, 10 gm. albumose | 17 | 13.5 | 15 | 257 |
| Pap of 80 gm. corn flour, 1 egg, 10 gm. sugar (two meals) | 7 | 5.5 | 90 | 398 |
| Total | 86 | 77.5 | 168 | 1711 |

III. *Milk Diet with Addition of Solid Food, Pastry, and Broths, leaving little Residue*

| | Albumin (per cent.). | Fat (per cent.). | Carbohydrates (per cent.). | Calories per 100. |
|---|---|---|---|---|
| Milk, 1250 cc. | 51 | 49 | 52 | 878 |
| Meat broth with 1 egg, 10 gm. of butter, 50 gm. of fine toasted wheat bread (or softened) | 10 | 14 | 30 | 294 |
| Cakes 70 gm., butter 15 gm. | 5 | 12 | 50 | 337 |
| Soup of 30 gm. tapioca flour, 1 egg, 10 gm. butter | 7 | 14 | 30 | 282 |
| Total | 73 | 89 | 162 | 1791 |

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[1] Ten gm. albumose is contained in 90 cc. (3 ounces) of Denayer's peptone preparation, in 22 gm. (℥vss) of Kemmerich's, or in 30 gm. (1 ounce) of Koch's.

IV. *Milk with Tender Meat, Solid Food (Pastry), Butter, and Soups*

| | Albumin (per cent.). | Fat (per cent.). | Carbohydrates (per cent.). | Calories per 100. |
|---|---|---|---|---|
| Spring chicken, 100 gm... | 19.6 | 2.8 | .... | 106.4 |
| Mashed potatoes, 100 gm.. | 2.0 | 4.0 | 20 | 127.4 |
| Two eggs.............. | 14.1 | 11.0 | .... | 160.1 |
| Toasted wheat bread 100 gm.................. | 7 | 0.5 | 55 | 258.8 |
| Butter, 30 gm........... | .... | 23.0 | .... | 213.9 |
| Trout, 100 gm........... | 19.3 | 2.1 | .... | 106.4 |
| Milk, 1250 cc. and soups in addition............... | 51 | 49 | 52 | 878 |
| Total............... | 113.0 | 92.4 | 127 | 1851 |

V. *Abundant Non-irritating Diet*

| | Albumin (per cent.). | Fat (per cent.). | Carbohydrates (per cent.). | Calories per 100. |
|---|---|---|---|---|
| Tender meat, 250 gm.[1].... | 49 | 7.0 | .... | 266 |
| Cacao, 20 gm............. | 4 | 6.0 | 8 | 105 |
| Three eggs[2].............. | 21 | 16.0 | .... | 235 |
| 100 gm. Zwieback........ | 8 | 1.0 | 75 | 349.4 |
| 100 gm. wheatbread...... | 7 | 0.5 | 55 | 258.75 |
| 50 gm. cakes........... | 4 | 2.3 | 36 | 187 |
| 50 gm. butter[3].......... | .... | 44.0 | .... | 407 |
| 40 gm. tapioca flour[4].... | .... | .... | 40 | 164 |
| 40 gm. corn flour (maizena)................ | .... | .... | 40 | 164 |
| 20 gm. sugar[5]........... | .... | .... | 20 | 82 |
| 1250 cc. milk[6]........... | 51 | 49 | 52 | 878 |
| Total............... | 144 | 125.8 | 326 | 3096.15 |

*Diet Scales.*—A convenient diet scale has been devised by Stuart Hart,[7] of New York. It is depicted in Fig. 98. The scale has a total capacity for 1000 grams, with subdivisions of 20 grams each. The dial can be rotated to any point desired, so that the zero point can be brought to correspond to the tip of the dial pointer. For example, suppose the food prescription consists of:

| | |
|---|---|
| Meat........................................ | 100 grams |
| Potatoes.................................... | 80 grams |
| String-beans................................ | 140 grams |
| Wheat bread................................. | 60 grams |

[1] Meat of various kinds, finely chopped, raw or broiled in butter, or roasted, cold or hot, given in two meals.

[2] Egg in cocoa, one in soup and one raw or soft boiled.

[3] Butter for starchy foods, soup, etc.

[4] Tapioca flour to thicken soup.

[5] Sugar for cocoa and cornmeal pudding.

[6] Milk for cocoa, pudding, and to drink.

[7] Jour. Amer. Med. Assoc., Aug. 7, 1909.

Place an empty plate on the scale and rotate the dial until the zero point is opposite the end of the pointer. Add meat until the pointer indicates 100 grams; then rotate the dial again until zero is opposite the pointer; then add potatoes until 80 grams is indicated, and so on, so that each time the zero point on the dial is brought opposite the pointer.

**Digestibility of Food.**—We may say that an article of diet is easily digestible if it makes small demand on the secretory and motor functions of the stomach, if it is readily absorbed, and causes no subjective disturbance. The scale of digestibility of foods has been arranged according to the length of time that they remain in the stomach, the motor and secretory functions in the healthy stomach acting together, and hence the length of time the material remains in the organ indicates its digestibility; good motor power for a definite food shows good digestion. In pathologic conditions the rule is not as absolute, for either secretory or motor functions may be perverted alone or together, and sometimes in opposite directions. Gastric digestion may also be vicariously assumed by the intestines.

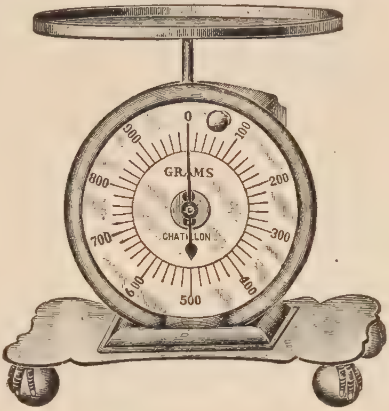

Fig. 98.—A food scale with an adjustable dial.

Leube's experiments were conducted with digestion in diseased stomachs, and from these he constructed his diet scale of foods according to their digestibility. It especially forms the basis of the diet of ulcer (Leube's method), and is valid in many other conditions.

**Leube's Diet Scale.**—*Diet* 1.—With much reduced digestion, the following are most easily digested: bouillon, meat solutions, such as Leube-Rosenthal's; milk; raw, soft-boiled, or poached eggs; zwieback; water, acidulous waters (Apollinaris, Seltzer).

*Diet* 2.—Less digestible are boiled calves' brain, boiled thymus, boiled chicken and pigeon, boiled calves' feet (with some); gruels, milk, mushes made with tapioca and beaten white of egg.

*Diet* 3.—If diet 2 is digested, then these can be given: Raw beef (chopped fine) or scraped meat with a dull spoon, and roast meat scrapings in fresh butter; raw ham (chopped fine); a little mashed potato; stale white bread and a small amount of coffee or tea with milk.

*Diet* 4.—Roast chicken; roast pigeon; venison; partridge; roast beef, medium to raw (particularly cold); veal (leg); pickerel; boiled shad; macaroni; bouillon with rice. Trout are hard to digest. Small quantities of wine one or two hours before eating. Gravies are contraindicated. Young and finely chopped spinach is best.

Asparagus may be tried, but Leube considers it risky. After the fourth diet the food is gradually increased, but very gradually. Vegetables, salads, preserves, and fruits should be refrained from for a long time. A baked apple is one of the first of these that can be taken.

Penzoldt has formulated a scale of digestibility for the normal stomach.

*Various Food-stuffs.*—It is important to remember that we can replace the albumin in food by carbohydrates, and at times by the fats, though the latter are not always well tolerated. In many cases the food should be concentrated, nourishing, and finely divided, so as not to irritate the organ.

Milk is excellent in some cases, but in ulcer, too large quantities, at least 3 liters (1770 calories), would have to be given, which tend to overdistend the organ.

Moreover, raw milk remains in the stomach longer than some other forms of nutriment, and when given alone, may coagulate, and with some disagrees. In some experiments at the Manhattan State Hospital,[1] in cases of dilatation of the stomach, it was conclusively shown that this organ emptied itself with greater rapidity in proportion to dilution of the milk with water. It is, therefore, preferable to give smaller quantities of milk and not to administer it alone, but diluted with barley-water, for example, or some other amylaceous material, such as rice-water or decoctions of tapioca, maizena, etc. One can add material such as raw eggs to increase the calorie value, a large amount of nutrient in small bulk, as described under the treatment of gastric ulcer. Gärtner's fat milk, from which some of the casein has been removed, is of service. Buttermilk has small nutritive value. Kefir, koumiss, bacillac, lactone, buttermilk, and matzoon are often useful.

Meat broth and bouillon have little nutritive value, while beef-tea and expressed beef-juice are of some service. It has been demonstrated, however, that bouillon or meat soup taken early in the meal soon reaches the pylorus and act as secretagogues, producing gastrin, inciting a flow of gastric juice. They aid thus in awakening dormant secretory glands.[2]

Beef-juice is best made from the rump of beef, cut in dice, cooked for five to ten minutes, and then the beef-juice expressed and pepper and salt added. Valentine's beef-juice and Armour's extract of beef can be recommended and are cheap.

Gelatinous articles of food, such as gelatin in solution, jellies, calves' feet, etc., are easily digested, as are soups containing chicken or calves' brain finely macerated and forced through a sieve. Gelatin does not produce indican, which is of great advantage.

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[1] Medical Record, June 20, 1908.

[2] The relation of the Food-stuffs to Alimentary Functions (Mendel), Amer. Jour. Med. Sci., Oct., 1909.

Shellfish, pike, halibut, and carp contain the least fat and are digestible.

Carbohydrates should be well masticated and carefully insalivated. If they contain much cellulose, they are not so readily digested and should be ground thoroughly. If there is stagnation and so danger of fermentation, care in their use should be exercised.

Ordinary bread (rye or domestic) is not so good. Zweiback, toast, maizena, tapioca, oatmeal, and Löfflund's Kindermehl are best.

Aleuronat flour (Ebstein) contains 80 per cent. albumin. Leguminose (Hartenstein's), Liebig's maltoleguminose, and Knorr's preparations are excellent. American veal has been found by experience to be not very digestible.

Fat in the form of butter (50 to 100 gm.) usually agrees. Fats inhibit gastric secretion, and this fact may be taken advantage of in the treatment of hypersecretion, and hyperchlorhydria.

Coffee and tea may be given in moderate amounts in many cases, but considerably diluted.

*Alcohol.*—Wines are recommended by many as a mild stimulant to the stomach in certain cases, but from personal experience I advise against their use. There are other remedies which give better results. The writer does not advocate the use of liquor in gastro-intestinal diseases. When stimulants are necessary, accurate dosage with cardiac stimulants he believes preferable.

*General Rules.*—The teeth should be kept in good condition; thorough mastication and insalivation are important, and a brief period of rest after meals should be advised both in health and disease. Regularity of meals should be enjoined.

*Method of Feeding to Spare the Stomach.*—In some cases it is necessary to administer nutriment in soluble or easily digested form. Appended is a short list:

Hemmerich's peptone, somatose, sanatogen, tropon, panopeptone, Koch's peptone, Brand's meat preparations, Valentine's meat juice, Liebig's soup, and Gärtner's fat milk.

*Leube-Rosenthal Meat Solution.*—Chop 1 kilo of beef into fine pieces, mixing it with water (1 liter), and add 20 gm. of pure hydrochloric acid and boil mixture ten to fifteen hours in a Papin pot. The mass obtained is crushed, boiled fifteen hours longer, neutralized with pure sodium carbonate, and evaporated to a mushy consistence. It is digestible, but contains relatively small quantities of peptone.

*Débove's Meat Powder.*—Roast finely chopped lean beef on tin plates until it is entirely desiccated. Powder in a mortar.

*Haggard.*—Cut lean meat into narrow strips, place for a few minutes in hot fat or lard until surface is browned, then place on a sieve for a short time. Fat is allowed to drip off, and meat is dried twenty-four hours in a baking oven at a moderate temperature. It can be ground to powder in a coffee-mill.

**Duodenal Alimentation.**—Einhorn[1] makes use of his duodenal pump for forced feeding. This instrument he employs for aspirating the duodenal contents, and for feeding simply reverses the process. The patient swallows a small perforated hollow bulb to which a thin tube is attached. After the bulb enters the duodenum, the instrument is left in situ for eight to twelve days, liquid nourishment being forced in every two hours from 7 A. M. to 9 P. M.

Analysis of results of three cases shows that case 1 left hospital with gain of ½ pound, but not free of complaint. Case 2 received medication by mouth during duodenal alimentation, and later mouth and duodenal feeding were both employed, and finally mouth-feeding combined with lavage. The case was ultimately cured of gastroptosis. Case 3, one of subacidity and nervous vomiting, lost one pound during duodenal alimentation, but vomited less, and on resumption of mouth-feeding the vomiting became more severe. No judgment can be formed from a neurotic case. In cases of ectasy with pyloric stenosis or spasm, the instrument often will not enter the duodenum; considerable pharyngeal irritation and discomfort are produced; it is difficult to force the liquid nourishment through the fine tube; there is no improvement in nutrition (gain in weight), and a foreign body the writer believes harmful in pyloric ulceration. Intelligent employment of nutritive enemata, combined with lavage, forced stomach-feeding (gavage), and posture on the right side after each feeding are indicated in cases of stenotic ectasy; the Lenhartz treatment in patients with gastric ulcer will improve the patient's condition and increase the weight. The writer *condemns absolutely* the *method of duodenal alimentation*, for the reasons given.

**Rectal Alimentation.**—*General Rules.*—The bowels should be emptied by enema; injection of the nutritive enema should be given with the patient on the left side, through a colon-tube several hours later; the temperature should be about 100° F. (warm). A folded towel should be pressed against the anus, and the buttocks pressed together for at least fifteen minutes after the injection.

*Milk should always be peptonized*, and alcohol, if injected, should not be stronger than 1 : 6 in the fluid enema. The addition of a small amount of salt aids absorption. Dextrose is readily absorbed. Occasionally it may be necessary to add a few drops of tincture of opium if there is much irritation. Raw eggs are readily absorbed. Sanatogen, peptone, and somatose are excellent additions. No more than 8 ounces (250 c.c.) should be given at an injection. It can be administered four or five times a day. If the bowel is irritable, smaller quantities should be employed. The patient should remain quiet for one-half hour after the injection.

Ewald suggests 3 to 5 raw eggs, mixed with 150 c.c. of water and 30 gm. of grape-sugar, and a small amount of common salt.

Boas uses 250 gm. milk; yolks of 2 eggs; salt, tablespoon of red wine; tablespoon of Kraftmehl.

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[1] Med. Record, July 16, 1910.

I have found the following useful: milk (peptonized), 125 c.c.; sanatogen, 3 drams (12); 2 raw eggs beaten up; water q. s. ad 250 c.c. (8 oz.), and a little salt. Modifications will readily suggest themselves.

*Anematose* (Fairchild).—This preparation, dispensed in one-ounce vials, consists of 60 per cent. by weight of pure nutritive solids. Its approximate composition comprises nitrogenous substances (beef and wheat), 12 per cent., wheat carbohydrates, 46 per cent., ash (phosphates, etc.), 2 per cent. It is bottled in sterile vials. Anematose is especially prepared for use as a nutritive enema, about 2 ounces of water being added before injection. Raw eggs and sanatogen may be added. The preparation is soluble and readily absorbed, and the author believes it a valuable adjunct when prolonged rectal alimentation is required. It is convenient for general use.

Boas[1] suggests the use of nutrient suppositories made of crystallized egg-white with a pinch of salt, dextrin, fat, and cocoa-butter. Each suppository contains 20 per cent. water, 2 per cent. salts, 20 per cent. fats, 33 per cent. carbohydrate, and 23 per cent. of protein, and has a value of 46.2 calories. He administers them four times a day. The nourishment seems insufficient to the writer, but the method may be tried if nutrient enemata cannot be retained.

*Leube's Meat Pancreas is Often Valuable.*—To 150 to 300 gm. of scraped and finely-chopped beef add 50 to 100 gm. of pancreas from cow or hog, free from fat, and finely chopped.

The two substances are placed in a dish, and 150 c.c. of lukewarm water are added, and the mixture stirred until it forms a thick, mushy mass.

If fat is also to be digested, add 25 to 50 gm. of fat. Inject with a pressure syringe.

Enemata of normal saline solution—1 dram (4.0) salt to 1 pint (500 c.c.) of water—are of value for thirst, or administered at a temperature of 110° to 120° F. as a stimulant. If this quantity is not retained, a smaller amount should be employed. Proctoclysis is useful. Hypodermoclysis with half to one pint of normal salt solution is of value to supply fluid to the body. Sterile almond oil, 1 dram (4 c.c.) might be given three times daily subcutaneously in extreme emaciation for a brief period.

Leube recommends as much as 50 to 100 gm. of bland sterile olive oil at a time, injected subcutaneously. As a rule the writer does not employ fats by this method. The subcutaneous use of peptones and albumoses is harmful, and the similar use of sugar is not advised.

Inunctions with preparations of oil or lanolin may at times be of service.

Gavage, or forced feeding through the stomach-tube, is referred to under Lavage through the nasal route.

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[1] Berlin. klin. Wochenschr., April 4, 1910.

## DIET IN DISEASE

General principles will be enumerated.

In *acute diseases of the stomach*, as in acute gastritis, the indication is to spare the organ as much as possible. In some cases no food is administered by mouth for several days and rectal feeding is employed. If food is administered, it should be given in small quantities at first and in liquid form—weak broths, bouillon, barley-water, a small quantity of water. They should be neither excessively hot nor cold. Peptonized milk, milk and lime-water, white of raw egg beaten up; later, toasted bread, soft-boiled eggs, etc., and a gradual return to full diet.

**Ulcer of the Stomach.**—In this condition both mechanic and chemic irritation of the stomach should be avoided. Albumin solutions and finely divided protein material are indicated, such as milk, beef-juice, somatose, tropon, or, *especially*, *sanatogen* in solution.

Einhorn allows barley-, oatmeal-, or rice-water in the early treatment. Raw eggs beaten up aid in binding the free acid. Dextrose and butter aid nutrition and lessen acidity. Starch in any great quantity is not well borne on account of the acidity.

Leube avoids all stomach feeding for several days after the hemorrhage, employing nutritive enema; while Lenhartz feeds immediately, binds the free acid, and endeavors to rapidly improve nutrition. *Gelatin solutions* are of value in these cases.

**Chronic Diseases of the Stomach.**—In the chronic diseases it is extremely important to see that sufficient quantity is taken and to improve the nutrition of the patient, as frequently subnutrition is present.

*Carcinoma.*—In malignant disease of the stomach or its orifices little can be accomplished by diet alone. The patient should receive frequent meals in small quantities, liquid or pultaceous in form. This is advisable, even if the cancer is so situated as not to interfere much with the motor function. I have seen cases improve temporarily in nutrition. Radical or palliative operation is indicated.

*Benign Stricture.*—Stricture of the cardia can at times be dilated with bougies, operation may be required; with pyloric stricture perfect recovery often follows operation. With benign pyloric stenosis, hyperacidity, at times with hypersecretion, and ectasia are present. The indications in these cases are to administer liquid or mushy foods, chiefly of albuminous type. There are relative motor insufficiency and increased secretion. Starchy food in quantity is not, therefore, well borne.

The gastric *secretion*, *motility*, and *sensibility* of the stomach must be studied in every case.

When there are *disturbances of the motor function*, *food should be given so prepared* that it will be *most easily evacuated from the stomach, as in liquid or pultaceous form.*

Among the disturbances of gastric secretion we have hyperacidity and hypersecretion, an increase in the secretory function; and subacidity and anacidity, an impairment of secretion. Motor disturbances may complicate any of these conditions, and sensory disturbances are most frequent in the first class.

In the hyperacid forms, a diet should be selected which stimulates hydrochloric acid secretion as little as possible and at the same time combines with it. Abundant albuminous diet should be administered, with hyperacidity, in coarse form if the motor function is intact, otherwise in liquid or mushy form. With hypersecretion, smaller and more frequent meals are indicated, and fluids should be limited. Carbohydrates in any quantity are not well borne, though their digestion is better with hyperacidity than with hypersecretion. Solutions of dextrose are readily absorbed and lessen the secretion of hydrochloric acid. The diet of ulcer has been previously referred to.

Fats are quite well borne, and in the form of olive oil lessen the hydrochloric acid secretion.

In cases of subacidity as in chronic gastritis, meat must be diminished and carbohydrate material increased. Nutrition must be improved. Koumiss, matzoon, milk, and raw eggs must be taken with crackers and butter between meals.

As an example of anacidity we have achylia gastrica. This may be a temporary functional disturbance or a permanent condition with organic changes. Chemic action has ceased and vegetable food (in which starch granules possess an albuminous coat) as well as animal food pass from the stomach unchanged and irritate the intestines. A rich carbohydrate diet is indicated, but it must be well pulverized.

With ectasia and gastroptosis small and frequent meals are indicated, the character depending on the gastric secretion. With severer forms of motor insufficiency liquids and mushes are required.

In nervous dyspepsia one must gradually increase the quantity of food. The administration of frequent small meals, koumiss, matzoon, etc., between the chief meals is of value. The rest cure is often serviceable.

One must individualize in every patient, and with the aid of the general principles described it will be possible to formulate a diet to suit the case.

**Methods of Feeding Before and After Gastro-enterostomy.**—This subject is of such importance that the author feels that he should offer some brief suggestions.

For a period of twenty-four to forty-eight hours before gastro-enterostomy the patient should be placed on a *sterile liquid diet*—broths, bouillon, milk, and water, all thoroughly sterile. The nares and pharynx should be sprayed with hydrogen peroxid, 20 per cent. solution, before and after each feeding. Acetozone, 5 grains daily, may be given in the sterile drinking-water in divided doses. The writer further advocates in addition to the usual examination of the urine, that a special examination[1] be made for acetone and diacetic acid, and if such be present, oatmeal gruel (strained) be given for several days previous to operation, and about 2 drams (8.0) soda bicarbonate be administered daily. Subsequent to operation, 3 drams (12.0) soda bicarbonate should be given daily in the nutritive enemata, thereby lessening the chance of vomiting due to acidosis. If chloroform is to be administered, preliminary alkaline treatment is useful even if no acetone be present before operation. Lavage should be performed about an hour before operation with acetozone solution 1 : 1000, the stomach being thoroughly washed and the contents well aspirated, so as to have an empty organ. If this procedure is not carried out, particularly if there is pyloric stenosis, there may be considerable fluid in the stomach, even though the patient has not been fed for twelve to eighteen hours previous to operation.

**Postoperative Feeding.**—The lips should be moistened with wet cloths after operation and proctoclysis or small enemata (4 ounces normal saline solution) be given for thirst. Four to six hours after operation nutritive enemata should be begun, and these may be given four or five in number during the twenty-four hours and so continued for a week after operation. Twelve hours after operation 1-dram (4.0) doses of hot water may be given by mouth at first at hour intervals and then increased gradually in quantity. At the end of twenty-four to forty-eight hours 1-dram (4.0) doses of strained barley water gruel or strained rice gruel may be given every hour, gradually increasing to larger quantities at greater intervals. On the third or fourth day peptonized milk in small quantities, well diluted with lime-water, and on the fifth to seventh days two to four raw eggs well beaten should be added.. Thereafter soft diet until the end of two weeks. Strained gruels the writer believes preferable at first to milk. The patients upon whom this operation is performed are usually in poor physical condition and proper methods of early feeding are indicated.

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[1] In view of the frequency of colon bacillus infection, the writer advocates examination of the urine for gram negative bacilli both before and after operation. If they are in evidence, cultures should be taken and treatment at once be begun. See chapter on "Infections by the Colon Bacillus." If indicanuria is marked, urotropin and lactic acid bacilli tablets should also be given preparatory to operation.

## CHAPTER VIII

# LOCAL TREATMENT OF THE STOMACH

### LAVAGE

SINCE the year 1867, when Kussmaul employed lavage in a scientific manner with his stomach-pump in the treatment of diseases of the stomach, there has been a gradual improvement in the type of instrument. The hard tube and the use of the stylet for introduction have passed into disuse, and the modern method is based upon the principle of siphonage with the soft-rubber tube.

**Funnel Method.**—The one that is in most common use for washing the stomach is by means of the funnel. The latter may be of hard rubber, glass, or a flexible rubber funnel, attached to a piece of soft-rubber tubing about a yard long, the latter being joined to the upper end of the stomach-tube by a small connecting glass or hard-rubber tube (Fig. 99).

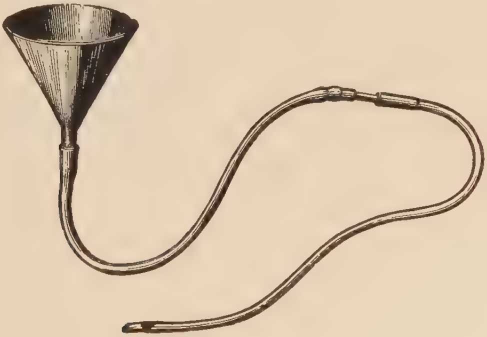

Fig. 99.—Funnel and tube for lavage.

The glass funnel is more readily broken and the soft-rubber funnel not as easily managed by a novice, so that for general use the hard-rubber funnel is preferable. One of medium size, holding about 250 c.c., is most convenient. The glass connecting tube, in one case to my knowledge, came near being the source of considerable danger to the patient. During the lavage he suddenly grasped the glass tube and managed to splinter off a portion of it, fortunately at the same time partially pulling out the stomach-tube. The accident was immediately noted and the tube entirely withdrawn. Fragments of glass were found in the tube.

There is always the possibility of a similar accident to the glass funnel, and the use of a rubber instrument and metal or rubber attachment would seem to be preferable.

*Dangers.*—There is one possible danger, namely, the stomach-tube may separate from the attachment to the funnel tube and slip down into the stomach. Leube[1] reports such a case. On the ninth day after swallowing the tube—after an attack of coughing—the tube passed up into the esophagus and pharynx and was withdrawn therefrom. The connection between the stomach-tube and funnel-tube had become loosened and the water from the latter forced the tube into the mouth.

Moreover, every stomach-tube *should be tested before using* to be sure that it is not cracked or weakened by some defect.

Friedenwald[2] reports such an accident resulting from the use a defective tube, with the result that two fragments were subsequently removed by gastrotomy.

At the Manhattan State Hospital, Ward's Island, among our nervous and insane patients, a long, continuous, single-piece stomach-tube (3½ to 4½ feet in length), with a rubber funnel at the end, is employed. This obviates all possible danger of swallowing the tube, which would be the most likely accident with this class of patients.

For ambulance work such a tube is decidedly to be recommended.

Under ordinary conditions, with the two-piece tube, the patient or *an assistant* can hold the stomach-tube tightly at the point of junction with the connecting-joint, or the operator can hold it firmly at the lips of the patient when he elevates the funnel.

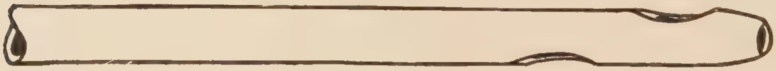

Fig. 100.—Best tube for lavage.

*Selection of the Stomach-tube.*—The selection of the stomach-tube may seem to be an unimportant matter, but from a varied experience there is undoubtedly a decided advantage in the choice of an instrument.

The tube should be of value both for aspiration of the stomach-contents and for lavage. Some prefer the tube closed at the end and with two lateral openings, claiming that there is less danger of traumatism from the smooth rounded end and less chance of aspirating the mucous membrane into the openings of the tube.

In actual practice, one has to exercise more pressure with this type of tube in order to force it to take the curvature of the stomach and lie parallel with the same, if thorough aspiration or lavage is to be performed. This tube is, moreover, not so readily cleansed.

The tube *open at the end* and with a large lateral opening is preferable; a tube with two lateral openings is the best, though not absolutely essential (Fig. 100).

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[1] Deutsch. Arch. f. klin. Med., vol. xxxiii, p. 6.

[2] American Medicine, August 2, 1902.

It is much easier to thoroughly aspirate the stomach or perform lavage with tubes of this description. The pressure is so minimized by these large openings that in my own experience I have never seen damage result to the mucous membrane.

For practical purposes a tube of from 28 to 30 (French) is thoroughly efficient. I have seen physicians in their general practice employ tubes of very large caliber, irrespective of the size or age of the patient. This is about as sensible as using a large sound in every case, irrespective of caliber or conditions of the urethra.

The tube of medium caliber that I have indicated will pass comfortably through any average or even small adult esophagus, unless stricture be present. The best tubes are marked at about 18 inches from the distal end (it is 16 inches from the teeth to the stomach), and by this means we know when the instrument has reached that organ. The tube should be inserted a little further until checked by the lower border of the stomach, and then withdrawn slightly until no resistance is felt by the operator. With ectasia or gastroptosis it may be necessary to pass it a considerable distance.

If the tube is not marked it is easy to estimate the correct distance and make a scratch-mark on the tube with a pencil, or insert it until checked by the lower border of stomach, as just described.

A pitcher, pail, or large basin, towels, and rubber sheets are required. Special irrigating stands can be secured, but are unnecessary and expensive for the young physician.

The correct method of lavage in office practice, without an assistant, is first described. *False teeth should be removed.*

The patient should sit upright in a straight back chair, with a rubber sheet or towels about the neck and protecting the front of the body. His confidence should be gained. He should be told that the procedure is slightly disagreeable, but absolutely safe. He should be admonished that he is to breathe deeply and steadily all the time, as this will prevent the gagging and sensation of choking; and he should be directed, while the lavage is actually in process, to follow out this method,

The stomach-tube should be lubricated by dipping it in warm water. Vaselin, cold cream, or olive oil may be used, but they are disagreeable and unnecessary. The irrigating fluid should be pleasantly warm to the hand. This is sufficient index to the temperature, though for absolute accuracy a thermometer may be employed; and about 100° to 101° F. is correct.

Plain water or, preferably, normal saline solution, 1 dram (4.0) of salt to 1 pint (500 c.c.) of water, is employed in the average case for the purpose of cleansing the stomach. Special solutions will be appropriately indicated in their places.

The patient should bend the head slightly forward and open the mouth, but not protrude the tongue.

In rare cases a 2 per cent. cocain or 5 per cent. eucain spray may be required if the pharynx is irritable. Freezing the stomach-tube is also serviceable. This consists in placing the tube for a brief period in ice or ice-cold water, so that it is extremely cold when it enters the pharynx, having an anesthetic effect.

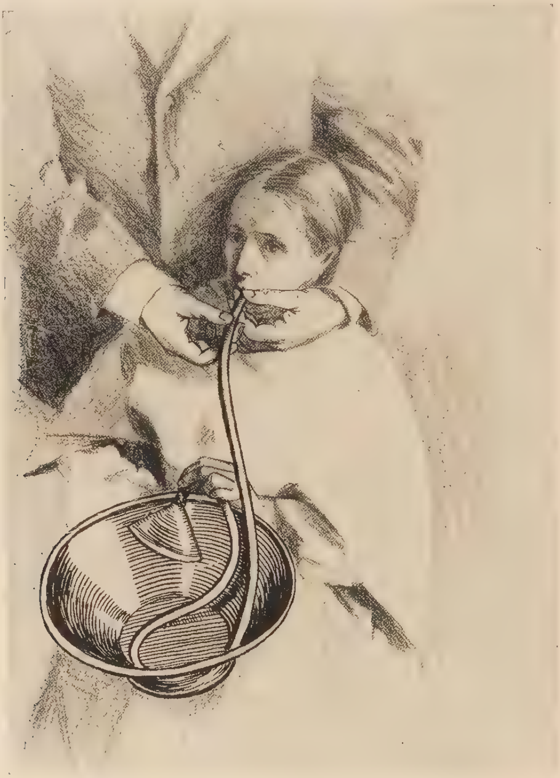

Fig. 101.—Correct method of passing the stomach-tube.

The physician should never insert his finger into the mouth to depress the tongue or act as a guide to the tube, as it renders the operation more difficult, and will only cause gagging or vomiting.

Most of our text-books advise that the operator stand in front of the patient and pass the tube along the base of the tongue.

The most practical method is the one shown in Fig. 101.

The physician stands on the right side and a little back of the patient and passes the left arm about the neck, the fingers *supporting the tube at the lips*, the little finger resting on the chin.

This method prevents the patient from throwing his head back and struggling, and gives the operator perfect control.

The stomach-tube should be passed into the mouth with the right hand, it being held about 2 inches from the lips, and being supported by the left hand against the roof of the mouth. It should then be rapidly forced in with the right hand, the index-finger and thumb of the left hand continuously aiding its introduction.

The tube follows the arch of the mouth, and glides down the posterior wall of the pharynx (Fig. 102). *Interference from the tongue is thus avoided.*

When its progress is checked, the patient should be told to swallow, and it will enter the esophagus. It should now be rapidly fed into the mouth until the marked ring has been reached, and then more slowly to the bottom of the stomach.

If the tube is checked during introduction, it is probably due to a spasm of the esophagus, and the act of swallowing or a deep inspiration will free it. There is practically no danger of its entrance into the larynx.

Sometimes the patient may become cyanotic, and a beginner may fear that this has happened; but this is due to the fact that the breath is held, and deep and regular breathing will immediately relieve the condition.

Occasionally the tube may slip out of the esophagus and coil in the mouth, but that is easily detected. Patients accustomed to lavage can often introduce the tube themselves.

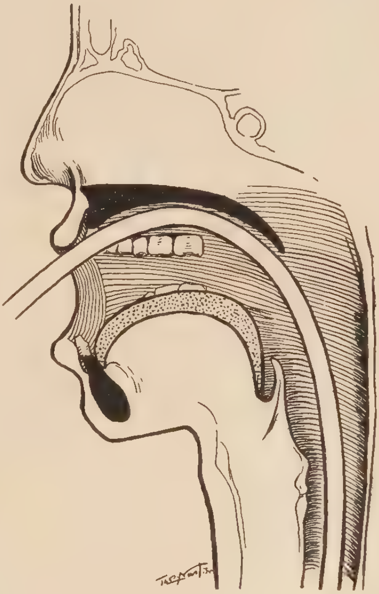

Fig. 102.—Course of tube.

I prefer a funnel[1] of a capacity of 250 c.c. or about 8 ounces, and allow 2 funnelfuls to run into the stomach (in all about 1 pint) and to run out again at once. It is a bad practice to overdistend the stomach, just as it is the bladder. At times I employ 1 funnelful.

Lavage is continued until the water is perfectly clear. The patient may move the body about so as to bring the water in more thorough contact with the stomach wall, or may lie down, as suggested by Fleiner, rotating to the right side and then to the left; but this is rarely necessary except in cases of marked dilatation with insufficiency.

*Lavage by a Single Operator.*—During lavage the patient steadies the stomach-tube, holding it to the lips with one hand. He should be instructed not to bite the tube.

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[1] If the funnel is of smaller capacity, more funnelfuls should be used; total 250 to 500 c.c. (8 ounces to 1 pint).

The operator holds the funnel in the left hand and pours the water into it from a pitcher in the right hand, then elevates the funnel to about the level of the patient's forehead (Fig. 103).

As soon as it is empty, he pours in the second funnelful. Then before the latter is empty he quickly lowers it to below the level of the patient's stomach, and allows the fluid to siphon out into a pail or bowl placed on the floor to the right of the patient (Fig. 104).

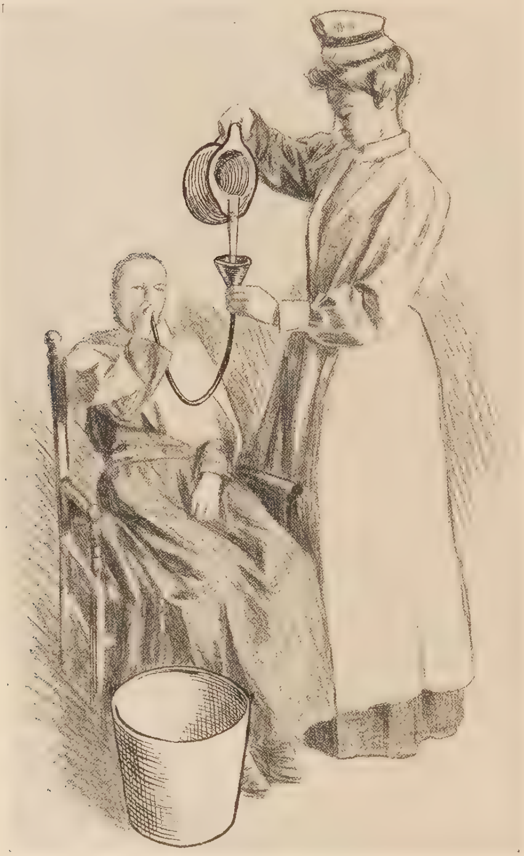

Fig. 103.—Lavage by single operator: Position one.

The rim of the funnel should be held upward. It should be allowed to fill before emptying it, as in this way it can be estimated whether the amount of fluid that flows out equals that which was poured in. In emptying the funnel it should only be slightly tipped, so that the column of water is still visible in the bottom. This prevents the entrance of air and also the aspiration of the mucous membrane into the openings of the tube.

When the washing is completed, the funnel should be rapidly raised and the stomach-tube withdrawn in this position. The small column of water remaining in the tube flows back into the stomach and prevents the possibility of the accident just noted. The tube should not be pinched during the withdrawal, as mucous membrane might be aspirated in.

If the water stops flowing during lavage, one should observe if the patient has not inadvertently withdrawn the tube slightly, and in this event, it should be pushed back.

On the other hand, the tube may be bent and the flow stopped by reason of its being forced too far into the stomach. If so, withdrawing it slightly will correct the trouble.

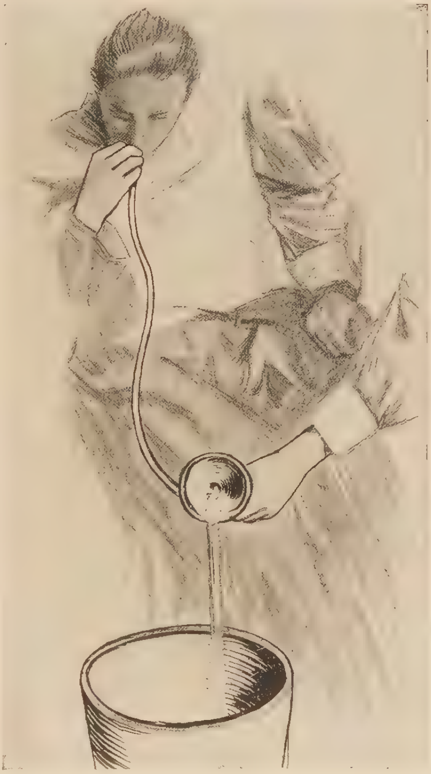

Fig. 104.—Lavage by single operator: Position two.

Pieces of food may occlude the tube by stopping up the stomach openings. By pouring more water into the funnel the instrument will usually be cleared out. By forcing air through the stomach-tube or by suction this can be accomplished if the above method fails. Thus, a rubber bulb with metal attachments is inserted between the stomach-tube and the funnel-tube, in place of the tube joining them. Close the end leading to the funnel and compress the bulb. This will force air through the stomach-tube; or first compress the bulb, then close the distal end connecting with the funnel by making an angle or compressing the soft tube of the latter, then let the rubber bulb expand again. The last method described is that originated by Friedlieb. The stiff rubber bulb made by Tiemann, with metal attachments, is a better instrument than Friedlieb's, which has glass attachments.

On rare occasions it may be necessary to remove the tube, clean it, and reintroduce it.

In emergency in country practice one can employ an ordinary tin kitchen funnel and a piece of small red or even white rubber tubing. A fountain syringe with the long soft-rubber tubing has been used in poison cases. A stomach-tube can be improvised out of a long colon tube. The lateral opening can be cut with a pair of sharp scissors, and the rough edges of the window burned over an alcohol lamp, wiping them quickly with a wet cloth, thus making a smooth velvet eye. If the opening of the improvised tube is rough, it can be trimmed down and smoothed by the above method.

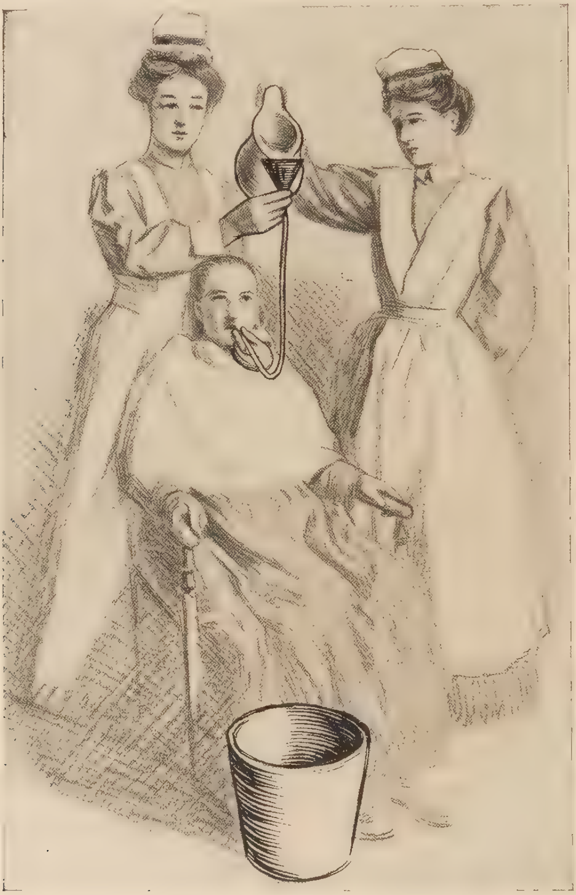
Fig. 105.—Lavage by two nurses: Step one.

It is best to lubricate all such emergency tubes with olive oil or vaselin, as they are less smooth and more difficult of introduction.

With an intelligent patient who will coöperate with the physician the method described is excellent. In nervous cases, when possible, it is easier to have an assistant or for two nurses to perform lavage, as in Figs. 105 and 106.

The technic is the same as to passage of the tube, etc. One nurse steadies the tube at the patient's mouth and carries on the manipulation with the funnel, the other pouring the fluid.

If the patient resist, nurse No. 1 both holds the tube and prevents interference by the patient's hands, while nurse No. 2 manages the funnel and pours the fluid. Friedlieb's apparatus is demonstrated in Fig. 107.

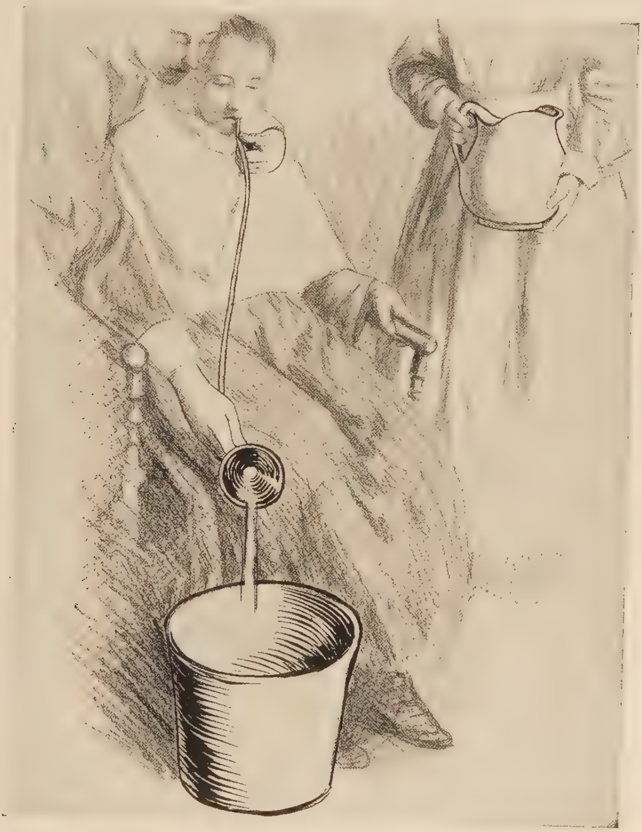

Fig. 106.—Lavage by two nurses: Step two.

In Figs. 108 and 109 we have two nurses performing lavage with a funnel, the *modified Friedlieb bulb* connecting the stomach-tube and funnel-tube. This is a favorite method of mine.

It is best to close the bulb on the side joining the stomach-tube by pinching the latter with the fingers, then squeeze out the air and pour the fluid into the funnel, allowing the bulb to fill with water. This prevents aspirating air into the stomach. Lavage is then performed in the usual way. The advantage is that we *have the bulb in position to employ air-pressure or suction* if the stomach-tube becomes occluded. Intermittent squeezing of the bulb also aids expulsion of the fluid.

The modified bulb made by Tiemann has metal connections (instead of glass) and is stiffer than Friedlieb's.

**Irrigation of the Stomach by Means of a Glass Y or T.**—This method has generally been known as Leube-Rosenthal's, but R. H. M. Dawbarn has taught this plan for many years. It is useful for office work.

A large glass irrigator, about two-quarts capacity, is hung at a level slightly above the patient's head. This should be marked in 250 to 2000 c.c. or in ounces or pints. This irrigator is connected with a long soft-rubber tube by means of a Y- or T-shaped glass or, *preferably, hard-rubber or metal* tube; one branch with the stomach-tube and the other with a carry-off tube which passes down into a pail or basin.

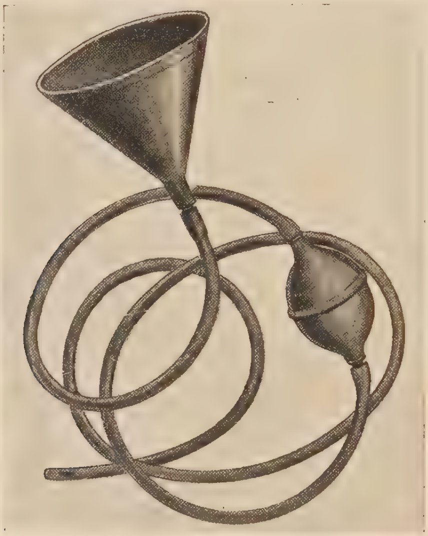

Fig. 107.—Friedlieb's apparatus for lavage.

The irrigator tube should be *closed with a clamp.* This is kept closed until after the introduction of the stomach-tube and commencement of lavage. A second clamp on the outflow tube is *unnecessary*, as all manipulation can be thereafter conducted by the operator, by alternately opening and closing the inflow and outflow tubes with the fingers.

The stomach-tube is introduced in the way described. The outflow is pinched as in Fig. 110, and the clamp on the inflow tube opened. Only 500 c.c. of fluid, or about 1 pint, is allowed to flow into the stomach. I do not approve of employing the larger quantities, as recommended by some authors.

While the fluid is still entering, the outflow tube is suddenly released and part of the current is diverted, thus starting the siphon action. The inflow tube is then pinched, as in Fig. 111, and the stomach rapidly empties itself. The outflow is then pinched and the inflow released, and so on. The patient may shake his abdomen, so as to wash all parts more thoroughly.

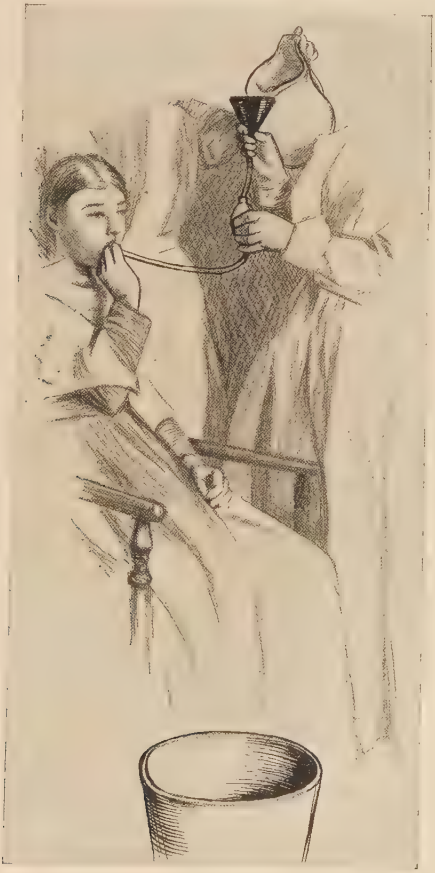

Fig. 108.—Nurses performing lavage with modified Friedlieb bulb: Step one.

This procedure must be continued until the wash-water returns clear.

There are cases among the insane or when the patient is unconscious when the methods described cannot be used.

It may be necessary to *employ a mouth-gag*, forcibly distend the jaws, and force in the stomach-tube. Under such conditions numerous assistants may be required.

The operation may be performed with the patient lying on the back, being properly restrained. The tube should be passed along the roof of the mouth, and the patient should lie on a sufficiently high level, so that the funnel can be carried below the level of the body in order to secure the siphon effect.

If no mouth-gag can be secured, the handle of a spoon, protected with gauze or a handkerchief, can be inserted between the teeth, turned, and the jaws forced apart, or some such instrument improvised.

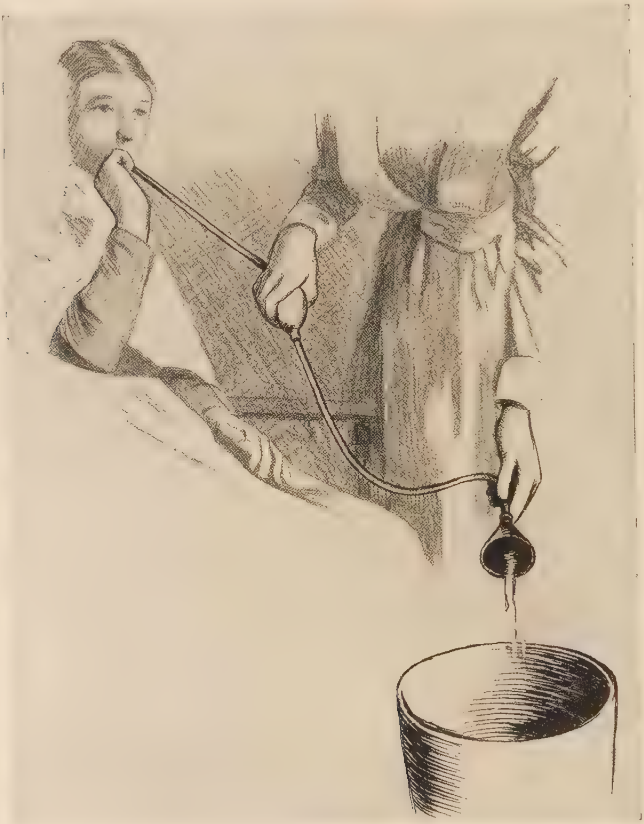

Fig. 109.—Nurses performing lavage with modified Friedlieb bulb: Step two.

The director devised by Mark Knapp (Fig. 112) would be of value in such cases. Being all metal, it can be left *in situ* during lavage, and acts as a gag.

The simplest method in such cases is to introduce the tube through the nostril.

The technic of lavage by this method is shown in Figs. 113 and 114.

The tube is passed along the floor of the nostril, just as is the guide for posterior nasal tamponade. It is fed directly through the nostril for about 20 inches, and no difficulty, as a rule, is experienced, as it passes down the posterior wall of the pharynx and so on into the esophagus.

The nares should be examined, and that nostril selected which is of larger dimensions. This is important, as there are frequently deflections of the septum

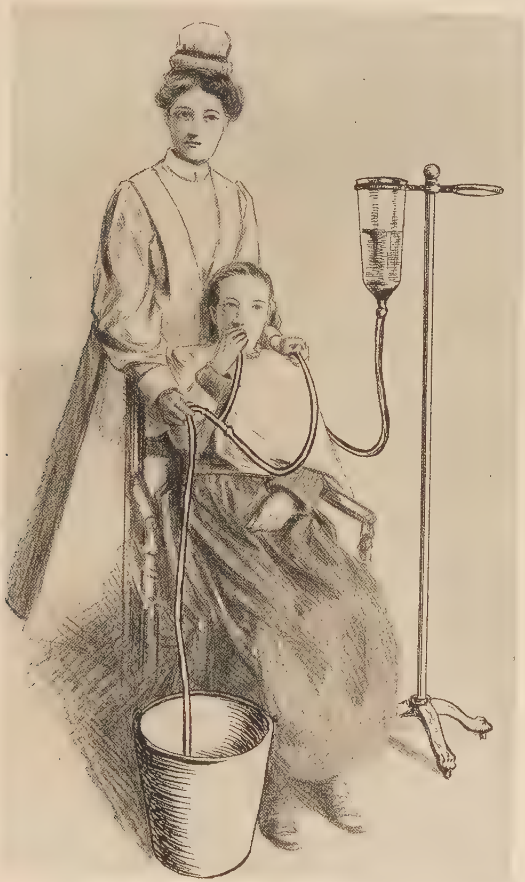

Fig. 110.—Stomach irrigation by the T-method: First step.

At the Manhattan State Hospital a stomach-tube of fair size (about 27, French) is employed. I frequently use this tube in my office lavage per orem. It is rarely necessary to employ the very small tubes so frequently recommended. For thorough lavage a fair caliber is preferable.

**Gavage.**—Gavage is a different proposition and the small tubes are to be preferred.

Gavage or forced feeding is employed chiefly among the insane who refuse food, the tube being introduced as in lavage, the *nasal route* being preferable. It may be necessary to employ this method of feeding in children who have been intubated and also in feeding the unconscious. In some cases, of course, the mouth route may be preferable.

Hemmeter and others have employed double-current tubes for lavage, but I can see no advantage.

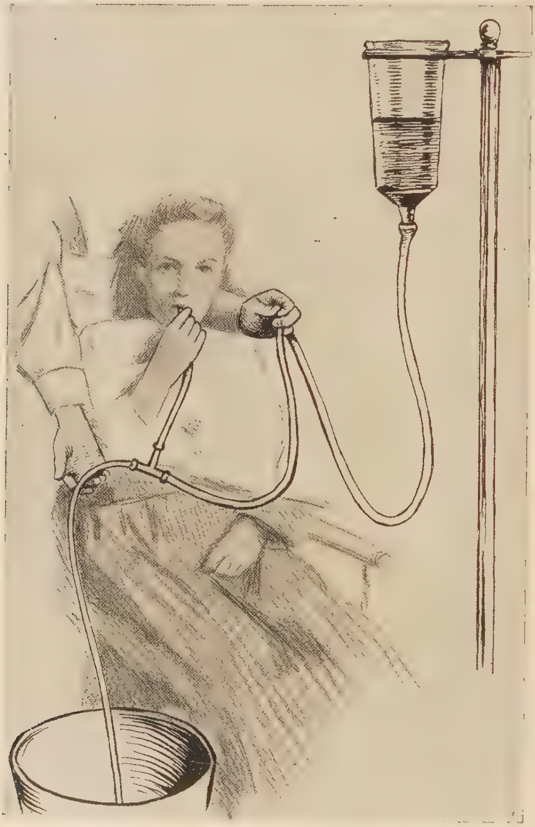

Fig. 111.—T-method: Second step.

**Indications for Lavage.**—1. *In All Cases of Poisoning.*—Some authors advise against it in cases from acids or alkalis, for fear of causing perforation; but there is greater danger of the latter by leaving the poison, since thorough emesis cannot be secured, especially if the patient be unconscious; there is the increased danger of subsequent damage to the intestinal canal below and often to the other organs, such as the liver and kidneys, or cardiac or respiratory poisoning might result.

2. In *acute* and *uncontrollable vomiting* from any cause, as from

acute gastritis, the value of lavage was first definitely shown by our specialists in pediatrics. Bilious vomiting is included.

3. In *chronic gastritis*, with excessive production of mucus.

4. In *dilatation of the stomach* (atonic type), where there are marked fermentation and motor insufficiency.

5. In *dilatation of the stomach* (stenotic type), with fermentation, motor insufficiency, gastritis, vomiting, etc.

6. In *acute dilatation of the stomach* from all causes.

7. In *vomiting due to vicious circle*, after gastro-enterostomy.

8. *At the end of anesthesia*, to prevent post-operative vomiting, or to treat the same, if it has occurred, before anesthesia, to prepare for gastro-enterostomy.

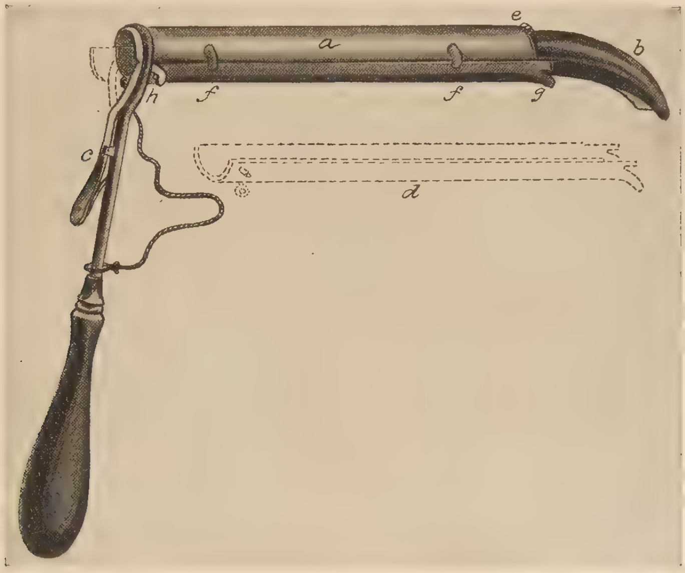

Fig. 112.—Knapp's director.

9. In *post-operative intestinal paresis* (correctly, gastro-intestinal paresis) lavage should be employed together with enteroclysis.

10. In *acute tympanitis of typhoid fever* lavage is of great value to aid reduction of the same; especially when hemorrhage is occurring and enteroclysis is contraindicated, see Typhoid Fever.

11. In *intestinal obstruction*, especially in intussusception. Frequent lavage has so relieved abdominal distention above the point of obstruction that the condition has been spontaneously reduced. It also checks the vomiting in this condition.

12. Occasionally lavage with iced water has proved successful in *uncontrollable hemorrhage from gastric ulcer*. It should be used as a last resort and with great caution.

13. It is also of service in *estimating the degree of motor insuffi-*

*ciency* by washing out the residue after aspiration following the test-meal.

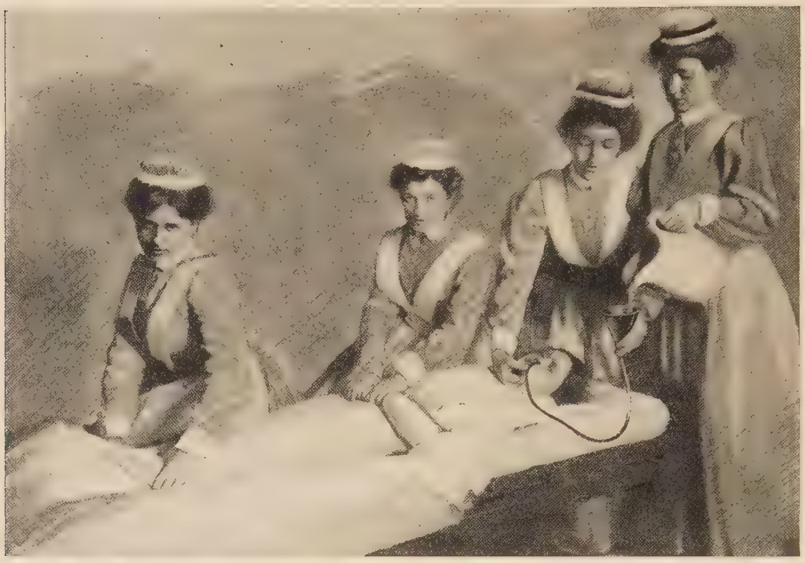

Fig. 113.—Lavage through the nostril: First step.

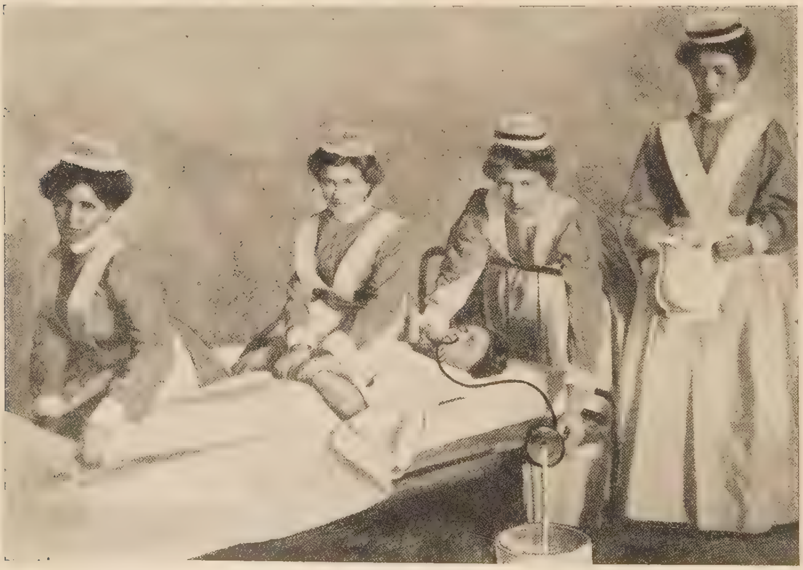

Fig. 114.—Lavage through the nostril: Second step.

14. In *gastric tetany*.
15. In the *convulsions of young children* which immediately follow

the ingestion of improper food. Epileptiform convulsions occasionally occur in adults from overfeeding and lavage is indicated.

16. In *vomiting of peritonitis.*

**Contraindications to Lavage.**—1. *Aneurysm of the aorta.*

2. *Gastric hemorrhage,* as a general rule, except as in No. 12 above.

3. *Marked heart lesions,* when danger might be incurred, such as in angina, etc.

4. *Last months of pregnancy.*

5. *Special conditions* where it might damage the patient or be dangerous.

In *cases of poisoning* lavage would take *precedence* over all other risks.

6. *Recent rectal, vesical,* or *renal hemorrhage.*

7. *Recent hemoptysis.*

8. *Extreme prostration* from any cause.

**Stomach Douche.**—This method was first described by Malbranc, and the measure was first employed by Kussmaul. It consists in sprinkling the stomach with water under pressure. Ewald and Rosenheim[1] have devised the most practical stomach-tubes for this purpose.

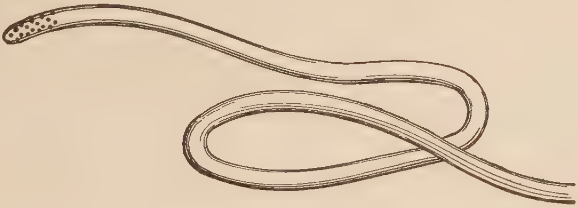

Fig. 115.—Tube for stomach douche.

The instrument, as in Fig. 115, has numerous small lateral openings and a slightly larger hole at the end. This last is so that the water can run off more rapidly in emptying the stomach, and any mucus or food products can more readily escape. If the hole is too large, the bulk of the fluid will pass through this and no diffuse irrigation of the mucous membrane be accomplished.

The tube is introduced in the manner already described, and the funnel method is employed. This is held quite high above the patient, so that the water is under considerable pressure and numerous small streams are forced out of the tube.

Rosenheim recommends it on an empty stomach before breakfast, or, if this is impractical, three or four hours after the first meal. It is applicable to mild motor insufficiency. He[2] advises its use in mild cases of chronic catarrh and in irritation of the sensory and secretory apparatus.

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[1] Therapeut. Monatssch., August, 1892.

[2] Berlin. Klinik, 1894, No. 71.

Salt added to the irrigation fluid increases the hydrochloric acid production, nitrate of silver (in 1 : 1000 strengths) reduces the secretion of gastric juice. Other observers have agreed with these findings, and Riegel advocates the use of the silver nitrate. Fleiner[1] recommends the douche for stimulating the appetite. Thus, infusions of hops, quassia, condurango, or cinchona bark are valuable.

Boric acid, salicylic acid, sodium salicylate, thymol, gomenol, creolin, lysol, etc., may be employed in average strength (1 : 1000) if antiseptic treatment is indicated.

If medicated douches are employed, the stomach should first be washed with plain water; then the medicated fluid applied for one to three minutes and then siphoned out; the stomach should be rewashed with plain water if toxic materials have been used.

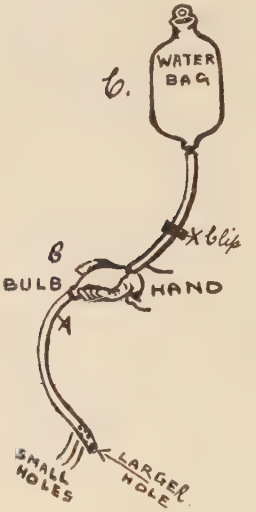

Fig. 116.—The bulb-compression method of spraying the stomach.

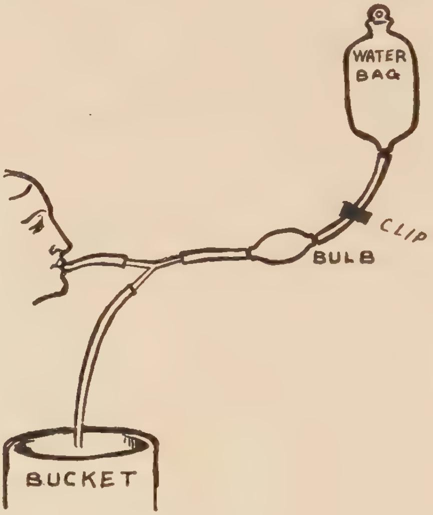

Fig. 117.—The Y-method of spraying the stomach.

Gross has devised a *double-current gastric douche*, which is scarcely practical, and Einhorn an instrument with a ball-valve and hard-rubber tip.

The following simple addition to the Ewald-Rosenheim tube gives satisfactory results. Employ a fountain syringe instead of a funnel and also a compression bulb (Fig. 116).

The stomach douche is passed in the usual way, and the bulb (aspirating) joins it to the fountain syringe, which contains the irrigating fluid at a temperature of 100° F. Clip X is previously closed. The tube is pinched tightly at *A*, and the clip then opened. By squeezing the bulb *B*, all air is driven out through the fountain syringe *C*. The bulb is then released and fills with fluid. This prevents air from entering the stomach. The finger releases the tube at *A* and the fluid begins to flow into the stomach; and by intermittent pressure of bulb (*B*) the spraying effect can be intensified at the will of the operator. When the douching is completed, the bulb is detached from the fountain syringe and with pressure of the same—the thumb over the end—slight aspiration is commenced and the contents will then siphon out.

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[1] Samml. klin. Vorträge, New Series, No. 103.

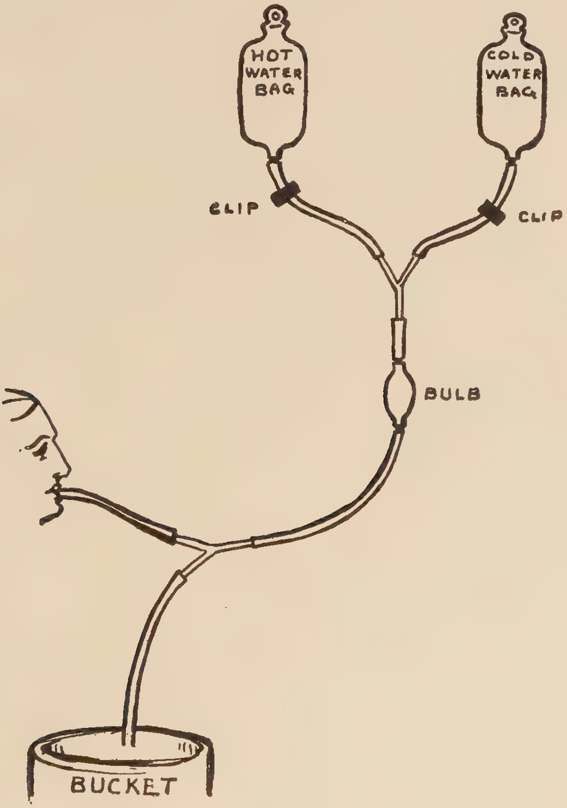

Fig. 118.—The alternate douche.

In Fig. 117 is illustrated the Y-method of spraying the stomach. The technic is the same as the similar procedure in lavage.

In Fig. 118 the use of the alternate hot and cold douche, with pressure bulb, is depicted.

The addition of the bulb enables one to employ the spray without distending the stomach with air, and to reach *much further than with simple hydrostatic pressure.* When stronger medicaments are employed, the rapid emptying of the stomach within one to two minutes and immediate lavage eliminate all danger, especially in view of the fact that by the compression method of spraying much less fluid is required, as compared with the older procedures.

The temperature of the fluid should be about 100° F. When stimulating effects are required, it could be 103° to 105° F. if cold, at 75° to 80° F. when used with the alternate douche.

In atony, with or without commencing dilatation, the spray method, employing in all not over $\frac{1}{2}$ pint to $1\frac{1}{2}$ pints, is at times of service to stimulate the organ. With this exception, I rarely use it.

**Gastric Spray.**—This consists of an ordinary spray apparatus with a double bulb, to which is attached a soft Nélaton tube 70 cm. long; within this is a fine central flexible tube, which connects the inner capillary tube with the nozzle (Fig. 119).

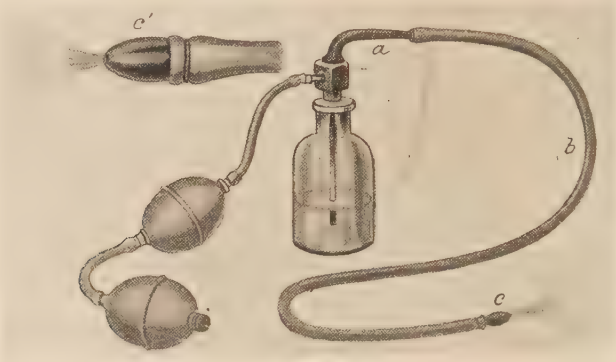

Fig. 119.—Einhorn's gastric spray.

Einhorn,[1] who devised the tube, recommends it highly, as thereby one employs a small amount of fluid to secure results; and hence there is less danger when toxic or irritating substances are used.

The spray should be employed when the patient has fasted or after a previous lavage. The bottle is filled with the required amount of the antiseptic solution, the tube dipped in warm water, and introduced in the usual manner.

The patient should hold the tube at the lips and the operator steady the bottle and compress the bulb. The spraying should be begun when the tube has entered to the mark (about 16 inches). It can be forced further in.

Einhorn advocates it to disinfect the mucous membrane of the stomach; for the application of astringents; and to relieve pain in gastralgia, as from ulcer, cicatrix, or cancer.

It is recommended in erosions of the stomach; in chronic gastritis, with marked production of mucus; in hypersecretion and hyperacidity, and in gastralgia.

Riegel holds that the necessary insufflation of air distends the stomach, and on this account it is objectionable. I have found the spray of value in gastralgia and in the treatment of erosions.

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[1] New York Medical Journal, Sept. 17, 1892.

### STOMACH POWDER-BLOWER

A dry method for spraying the stomach with insoluble substances (powders) has been devised by Einhorn.[1] His instrument consists of a flexible rubber tube about 28 inches long (Fig. 120), the distal end of which connects with an air suction-bulb.

The extremity of the tube is attached to a hard-rubber piece *c*, which is hollow and has lateral openings. It is provided with a screw thread. To this is attached a capsule with numerous side holes. Capsules of several sizes are furnished. A capsule is filled with powder and screwed on to the tip piece. A small spoon is employed to fill the capsule.

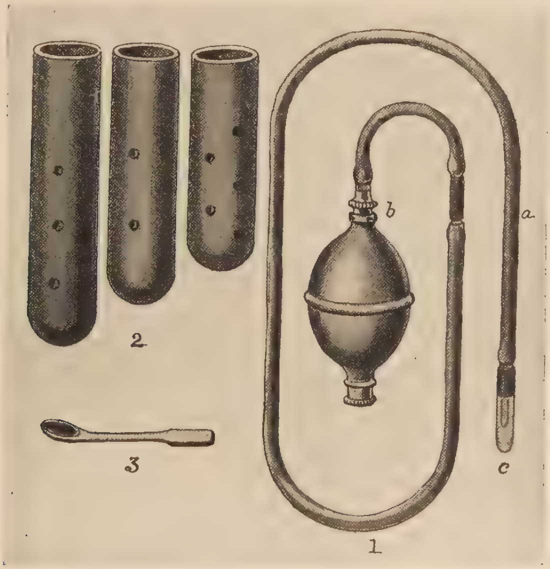

Fig. 120.—1, The stomach powder-blower: *a*, the tubing part; *b*, connection with the bulb; *c*, hard-rubber end with screw thread for capsule; 2, the capsule-shaped powder receptacles (natural size); 3, the small spoon for putting the powder into the capsule.

It is well to lubricate the latter with a thin layer of vaselin to prevent entrance of moisture. The tube is then dropped into warm water and inserted into the stomach in the usual manner. The bulb is quickly compressed several times and the air drives out the powder, opening up the vaselin layer over the holes.

It has been recommended for ulcer of the stomach, employing bismuth subnitrate; in gastralgia, orthoform; and in erosions, protargol or suprarenal powder.

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[1] New York Medical Journal, April 1, 1899.

I would not care to advocate the passage of the instrument in ulcer, but in the other conditions it is at times of service. Recently Einhorn has modified this instrument (Fig. 121). There is a double bulb with a stop-cock. This last is closed. Bulb *a* is compressed several times, thus overdistending bulb *b*, which is without valves. The stop-cock is opened and a single blast of air distributes the powder: about 15 gr. (1.0) is the capacity of the capsule.

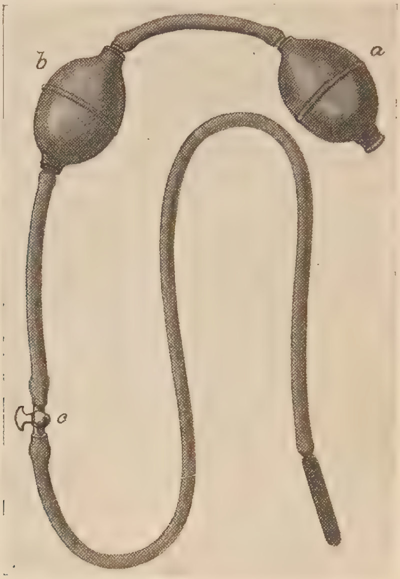

Fig. 121.—New model powder-blower.

## ELECTRICITY

From clinical experience it is found that the electric current exercises an influence on the secretory and motor functions of the stomach and also on its sensibility. Physiologic experiments and clinical experience do not always agree.

Meltzer, experimenting on animals, passed strong induced currents through the fundus of the organ and noted no contraction of the pylorus. The influence of the anesthetic or of morphin or similar drugs would influence the experiment.

Pepper demonstrated on a very thin patient that percutaneous electricity produced no peristaltic movements in the stomach. It is believed that it is through contraction of the abdominal muscles that this procedure influences the musculature of the stomach. The two methods for the application of the galvanic and faradic current are the percutaneous and the intraventricular. The *latter seems preferable from a therapeutic standpoint*, but the former is easier and is more readily submitted to by the patient.

**Percutaneous Method.**—Von Ziemssen employs two large plate-electrodes, one anteriorly between the pylorus and fundus, and the other from the fundus to spinal column, with a separating space of only 2 cm. The electrodes should be moistened, and sufficient current employed to cause strong contraction of the abdominal muscles. If smaller sponges are used, they can be moved about in these regions. The séance should last ten to fifteen minutes, and be carried out at first every other day, depending upon indications, which are the same as for the intragastric method.

**Intragastric Method.**—Kussmaul first suggested the internal application of electricity, and was the first to introduce the sound with a copper wire and olive point into the stomach. Bardet improved upon this, and employed an electrode which did not touch the stomach wall, the circuit being established by filling the organ with water.

Numerous intragastric electrodes have been devised, of which the most practical are Einhorn's, Lockwood's, and Bassler's.

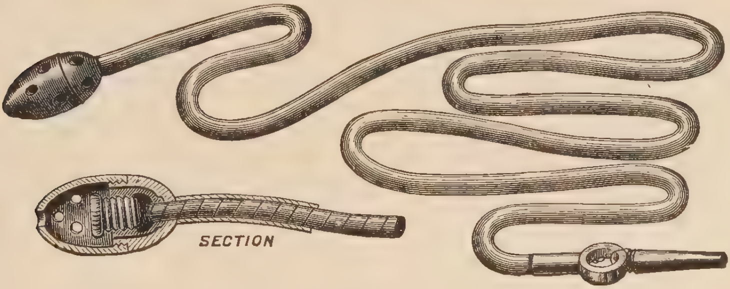

Fig. 122.—Lockwood's intragastric electrode.

**Lockwood's Electrode.**—This consists of a very small cable of conducting wire covered with rubber. The intragastric tip is olive pointed and protected from the gastric mucous membrane by a rubber fenestrated capsule (Fig. 122). The instrument in appearance is much like his gastrodiaphane. It is easy of introduction and of such small caliber that it does not incommode the patient. A glass or two of water is administered on the empty stomach, and the instrument is passed along the roof of the mouth, like the stomach-tube, about 18 inches or, preferably, until the resistance of the stomach wall is encountered. It is then slightly withdrawn. The outer end of the instrument is furnished with a key which is inserted into the negative pole of the battery. The sponge which is connected to the positive pole is applied over the stomach.

**Einhorn's Deglutible Electrode.**—The intragastric tip is similar in construction to Lockwood's. The conducting wire is much more flexible than Lockwood's and is covered with thin rubber. It is not introduced by the operator, but swallowed by the patient, being similar in principle to the stomach bucket (Fig. 123).

The key is connected to the cord of the negative pole of the battery. The patient drinks about a glass of water. The deglutible electrode is placed on the root of the tongue, and the patient is directed to swallow more water, which carries the instrument into the stomach. A mark can be placed on the cord, about 20 inches from the electrode, to determine that it has reached the bottom of the stomach. The sponge is attached to the positive pole. If there is resistance to the withdrawal of the instrument, the patient should swallow to relieve the spasm.

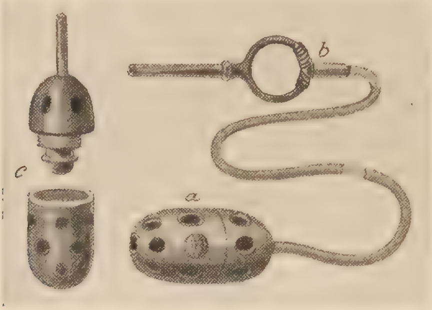

Fig. 123.—The deglutible stomach electrode.

**Bassler's Gastric Electrode.**—This instrument is practically a combination of Figs. 122 and 123. It has the usual capsule with a cord-like conducting wire and also an introducer (Fig. 124).

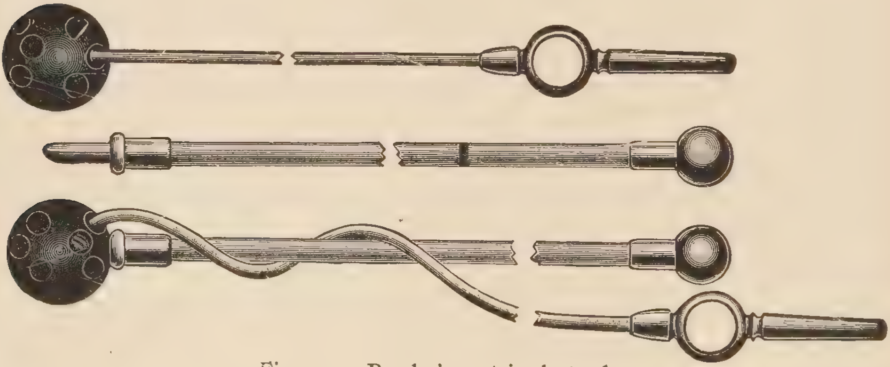

Fig. 124.—Bassler's gastric electrode.

The latter is withdrawn after the introduction of the electrode. After the application is completed the electrode is removed by the flexible conducting cord.

I have found Lockwood's electrode easy to introduce and unobjectionable to the patient. All are good instruments.

**Gastrofaradization.**—Duration ten to twelve minutes. The intragastric electrode is attached to the negative pole. A plate electrode connected with the positive pole is placed in the epigastric region for four or five minutes, and later a sponge. The electrode is then moved from left to right several times in the gastric region; and later, if marked constipation, from the caput coli to the sigmoid along the colon, and also over the umbilical region. The application over the stomach occupies about two minutes. The electrode is then placed to the left of the seventh dorsal vertebra for one to two minutes, and then returned to the front for the balance of the time. The current should be strong enough to cause contraction of the muscles, *but not produce pain.* Application of the sponge to the intestines occupies several minutes, in addition to the ten minutes over the stomach. I sometimes reverse the poles during the application, by changing their external attachments to the battery.

**Gastrogalvanization.**—Duration, eight to ten minutes. A small sponge electrode is placed on the epigastrium. The intragastric electrode is connected with the negative pole. The strength of the current should average 15 to 25 milliamperes. The full strength is not used at first. The application is two minutes to the epigastrium. It is then moved about over the gastric region for three to four minutes; then for one to two minutes in the dorsal region as described, and the balance of the period in the gastric region.

There has been some dispute as to the effects of these currents and the indications for their use. In general we may say that direct faradization increases the gastric secretion, while galvanization decreases it.

Faradization affects the musculature and galvanization the sensory field. The claims regarding increase in absorptive power from the use of electricity seem hardly to be substantiated, as it was so small as to be within normal limits.

**Therapeutics.**—The percutaneous method has proved of service in nervous anorexia and in motor insufficiency. The intragastric method is preferable when possible. It renders some patients more nervous, and in such should be avoided.

Among the indications for gastrofaradization are: Atonic ectasia, atony, relaxation of the cardia and pylorus, and diminished secretion.

For gastrogalvanization they are: Gastralgias, especially of nervous type, nervous anorexia, and hyperacidity.

Faradization, however, has proved of benefit in gastric neuroses.

**Triphase Method.**—Hershell[1] recommends the polyphase or triphase method in which two electrodes are placed on the back and one on the abdomen or intragastrically.

**Static Electricity.**—This has been recommended especially in the *atonic types* of ectasia, claim being made that it causes contraction of the organ. In neuroses it is sometimes of service.

Tousey[2] states that the motor functions are favorably influenced by the static indirect current.

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[1] Archives of the Roentgen Rays, 1906, 1907, vol. xxi, p. 221.

[2] Medical Electricity and Roentgen Rays, p. 400.

**High-frequency Current.**—This method is scarcely applicable by the general practitioner, but I believe it of some benefit in gastric neuroses, in atony, and for hyperchlorhydria.

**Türck's Gyromele.**—Türck has introduced this instrument for the local treatment of the stomach and colon. It consists of a cable with a sponge attachment, which can be made to revolve within an outer stomach-tube. There is an arrangement so that medicated fluids can flow into the stomach or colon through the outer tube, and also an attachment for a battery pole. He advocates its use for catarrhal gastritis to cleanse the mucous membrane. It can also be employed alone for internal massage of the stomach or combined with electricity. It is depicted in Fig. 125. The author questions its utility.

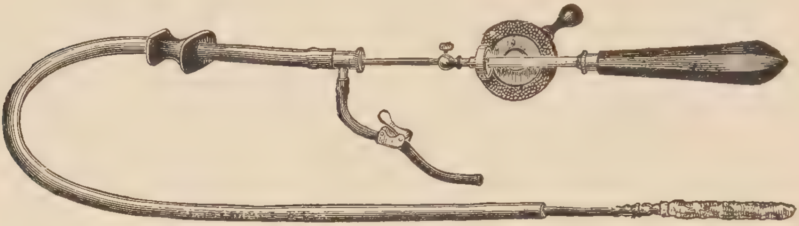

Fig. 125.—Türck's latest gyromele.

## CHAPTER IX

# MASSAGE—VIBRATORY MASSAGE—HYDROTHERAPY—COUNTERIRRITATION—ORTHOPEDIC APPLIANCES

### MASSAGE

MASSAGE of the stomach is indicated in atony or in the atonic form of dilatation and temporarily to aid in the removal of gas. The intestines should also be manipulated in these conditions. It is of value in stimulating the abdominal muscles in gastroptosis. I will briefly refer to a few simple methods.

If massage is performed on the absolutely empty stomach, it is contracted and cannot be palpated. It is preferable to perform it two or three hours after a meal. This aids in emptying the stomach. Earlier manipulation might cause vomiting.

Contraindications are ulcer, recent hemorrhage, and acute inflammation.

The patient should be in the dorsal position; lower limbs flexed. The left hand of the operator is placed on the right hypochondrium to exert counterpressure against the pyloric end. With the thumb and fingers extended, the right hand performs stroking motions from left to right over the stomach. Then the stomach is kneaded. These procedures should alternate. This technic should be carried out daily for five to ten minutes. With dilatation or ptosis of the stomach the direction of the stroking must be adapted to the position of the organ in each case.

**Tapping** (tapotement) or rapid vibratory movements with the fingers can be employed. It is often well to rotate the patient to the right side during massage, so as to aid in emptying the atonic stomach.

### VIBRATORY MASSAGE

Various vibrators, especially electric, many of which are quite expensive, have been devised for this purpose. Of late, vibrators can be purchased at a reasonable figure which can be attached to the street current[1] in the patient's residence. The method is very convenient when the facilities exist. There is an instrument run by carbonic acid gas pressure, which necessitates carrying a large tank. There is a small portable vibrator, the Vedee, manipulated by hand (Fig. 126), which is cheap, simple of manipulation, and efficacious. The strength of the vibration is regulated by changing the position of the rotating disk. The instrument can be employed, with the addition of electricity, by attaching a sponge arranged for battery

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[1] The Eureka vibrator is convenient, having many excellent attachments.

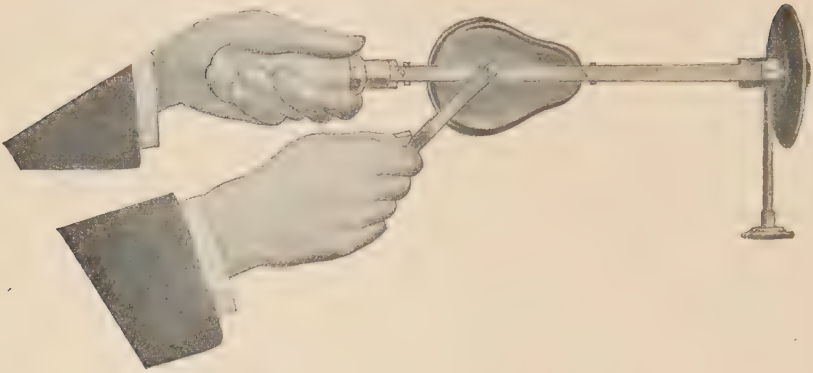

Fig. 126.—Vedee vibrator (new model).

connection and employing the other pole with a sponge over the

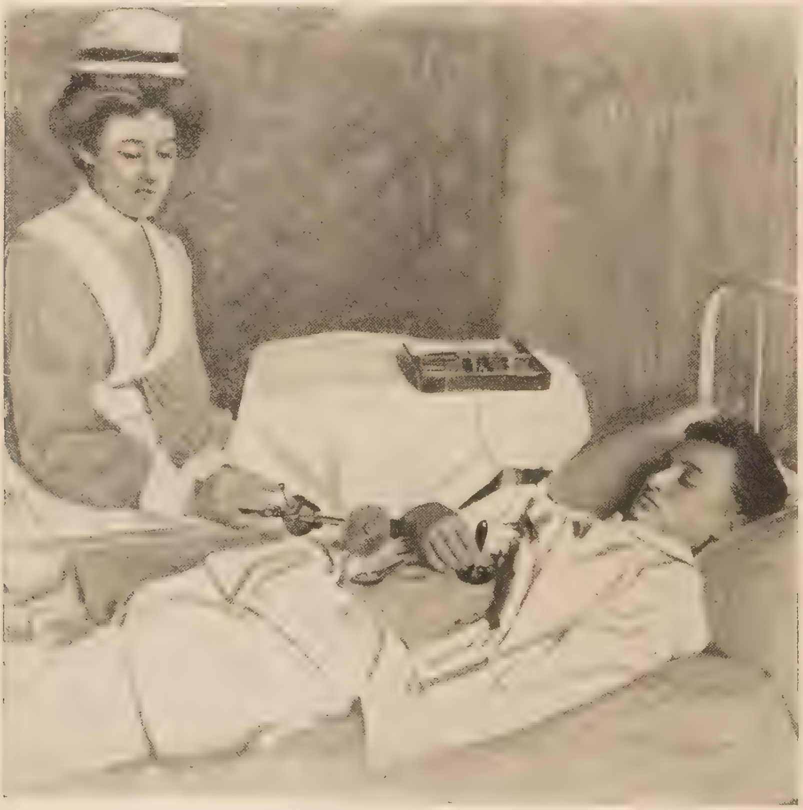

Fig. 127.—Combined electricity and vibratory massage.

abdomen. Electric vibratory massage can thus be given over the stomach and intestines (Fig. 127). In addition, one battery pole can be attached to the vibrator and the other to the intragastric electrode for treatment of atony of the stomach.

There are other small hand vibrators now manufactured and also small vibrators which can be run by a portable storage battery.

Vibratory massage should be given from left to right over the stomach for about three minutes, then two minutes to the left of the seventh dorsal vertebra, and three minutes more over the stomach. This should be performed daily.

It is generally advisable to vibrate the intestines, following the course of the colon, especially over the sigmoid, and also over the small intestines. This last can be done before breakfast to stimulate the bowels. Two or three hours later the stomach should be vibrated. The patient's family can be taught to use this instrument.

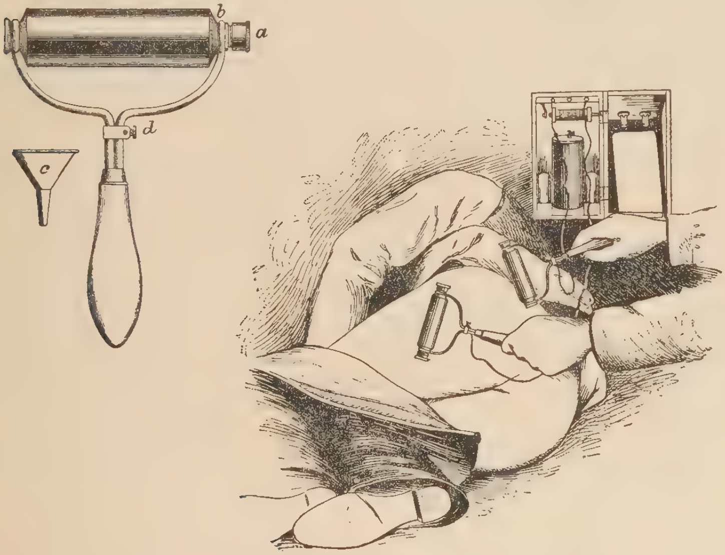

Fig. 128.—Massage roller with demonstration of alternating hot and cold electric massage: *a*, Screw cap; *b*, filling tube; *c*, funnel for filling; *d*, attachment for battery pole.

Bassler has recently devised an excellent vibrator, but it requires the street current.

**Massage Roller.**—Various instruments have been devised, but the following is simple. It consists of a revolving cylinder, which can be filled with hot or cold water, and which is furnished with a battery attachment. Heat or cold, or with an instrument attached to each battery pole, alternating heat and cold, combined with electricity, can be applied (Fig. 128). This method can be employed for abdominal massage in some cases of chronic constipation.

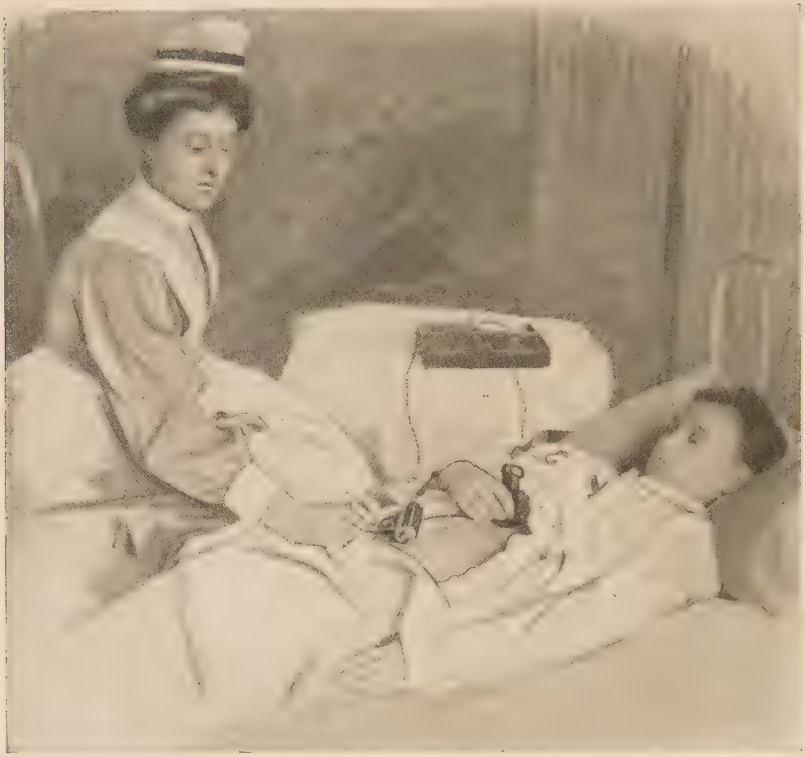

Fig. 129.—Combined application of electricity and roller massage with heat.

I have found the method next depicted of some value in stimulating atonic conditions of the gastro-intestinal tract (Fig. 129).

## LOCAL HYDROTHERAPY

**Cold.**—For hemorrhage or acute inflammation, the ice-bag is preferable to the Leiter coil. At times it relieves ulcer pain more than does heat.

**Priessnitz's Compress.**—A towel folded several times is dipped in cold or warm water, then wrung out, and placed over the stomach. Oiled silk or gutta-percha is placed over it, and a flannel binder applied to keep it in place. A temperature of 50° to 75° F. or warmer if desired can be employed.

This method is of value in nearly all painful diseases of the stomach. The compress can be changed two or three times a day. Some patients do better with the cold, others with the warm compress.

**Hot Applications.**—*Moist Heat.*—For cardialgia, ulcer, vomiting, etc., hot moist applications are of value. Poultices can be made of linseed, flaxseed, or bran, boiled in water, or of bread and milk. I have seen hot meal or hot mashed potatoes used in country practice.

The poultice is wrapped in gauze or cheese-cloth and applied as hot as the patient can bear it. Fresh hot poultices are continually applied. There is an apparatus which can be boiled in water, wrapped in a cloth, and then applied over the cataplasm. It will keep it constantly hot.

A felt sponge dipped in boiling water, wrung out, and covered with oiled silk can be employed.

**Dry Heat.**—The hot-water bag, hot cloths, a light tin pie-plate, heated in the oven and covered with flannel, the Japanese hot box containing burning punk, are all useful. A continuous hot-water coil has been devised, to be connected with a faucet.

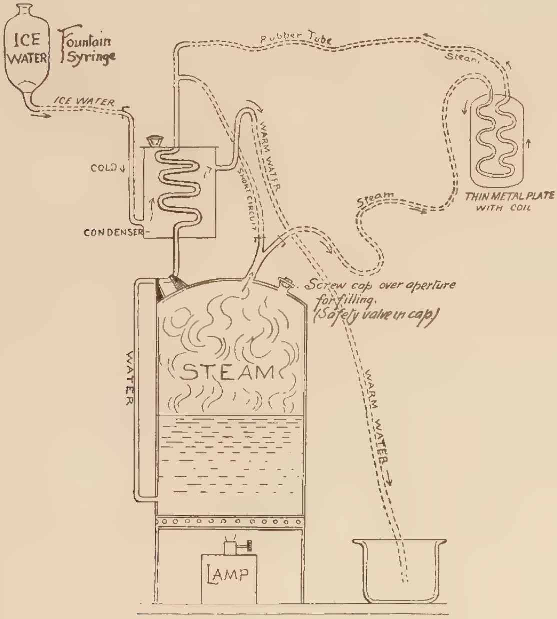

Fig. 130.—Continuous steam coil for the application of heat.

In the illustration (Fig. 130) is shown a continuous steam coil[1] of my own. The steam passes through a metal coil plate and is recondensed. Only a small quantity of water is necessary in the boiler. The temperature can be regulated by the coverings of the plate and by the stop-cocks on the Y-branch at the top of the boiler, thus allowing less steam to enter the coil. The coil can be placed over a moist poultice to preserve its heat. In general, moist compresses are preferable.

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[1] Enteroclysis, Hypodermoclysis, and Infusion, 1900.

**The Douche.**—The fan-douche and the Scotch douche, played over the stomach region, alternating cold 55° F. and warm 95° F. for about three minutes, may have a tonic effect. Packs, rubs, baths, and the carbonated bath are at times employed for the general tonic effect.

## COUNTERIRRITATION

Mustard and flour poultice (equal parts), or black or red pepper, 1 dram (4.0) to the pint (500 c.c.) of boiling water, flannel to be wet therein, wrung out, and applied with an oiled silk cover or a turpentine stupe, prepared by the same method, are of service.

## ORTHOPEDIC METHODS

Mechanical support of the stomach and intestines is of great service. Many nervous symptoms referred to the gastro-intestinal tract can be imputed to ptosis of the viscera and relaxation of the abdominal walls. In simple atony of this tract the proper support frequently aids the bowel action.

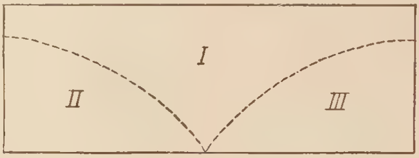

Fig. 131.—Pattern for cutting the Rose plaster abdominal binder: Dotted lines for section.

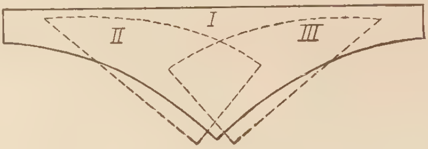

Fig. 132.—Pattern for cutting the Rose plaster abdominal binder: Plaster after section.

The indications for mechanical support are as follows:

*Ptosis of any or all of the viscera from any cause;* atony of the stomach; atonic dilatation of the stomach; stenotic ectasia as a temporary measure; atony of the intestines; pains from intra-abdominal adhesions dragging on the viscera; hernia of the abdominal wall; after laparotomy as a temporary support; postpartum to prevent ptosis (Landau's disease), and also to enable the patient to sit up in bed earlier and so drain the uterus. This last was suggested by me to Douglas H. Stewart, who has reported successful results. Cases of constipation in which atony is a factor. Pelvic disturbances which are associated with splanchnoptosis and result from general prolapse. Mucous colic in which ptosis is a factor. Pendulous abdomen, vomiting of pertussis (T. W. Kilmer), seasickness, and nervous vomiting.

There are three methods: The use of adhesive plaster, the most scientific; elastic bandages, and special corsets.

To A. Rose we must credit the best method of adhesive strapping. The author first suggested the use of zinc oxid on moleskin plaster, from experiments finding it most suitable, and this was adopted by the originator of the method. For a full description I would refer to our work on this subject.[1]

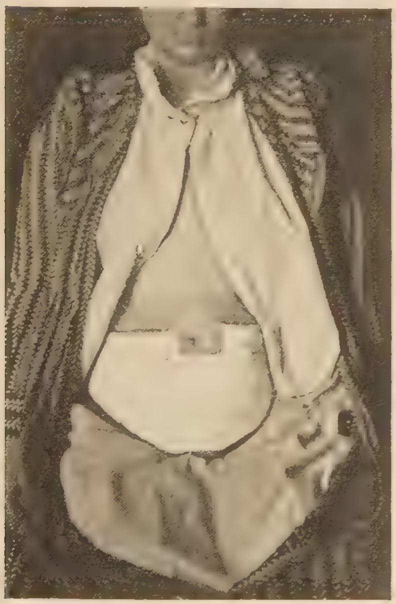

Fig. 133.—Rose's belt (under plaster): Step one.

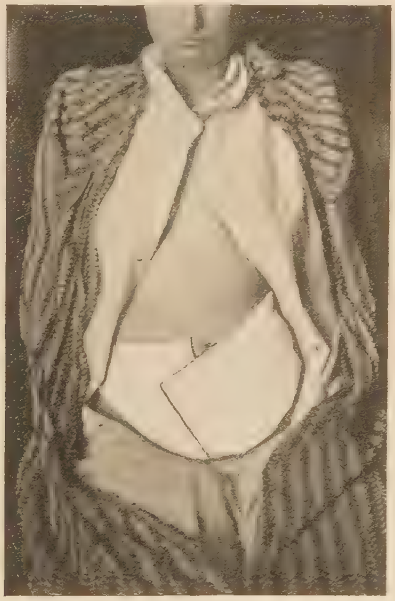

Fig. 134.—Rose's belt: Step two, left wing.

The method is as follows: Adhesive plaster, zinc oxid on soft moleskin (Johnson and Johnson), preferably 7 inches wide, though 6 inches can be employed. A yard in length will suffice in most cases. The circumference of each patient should be measured, and the plaster should be long enough to encircle the waist. The plaster is folded over so that the free ends are in line and a curved line drawn in pencil from the lower margin of the point where it folds to the free margin, to about 1 inch below the upper border. The plaster is cut along this line, giving three pieces: or the plaster is stretched out and the dotted lines marked, as in Fig. 131, and cut along these lines, giving three pieces, *I* and the two lateral pieces, *II*, *III*, as in Fig. 132.

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[1] Rose and Kemp, Atonia Gastrica.

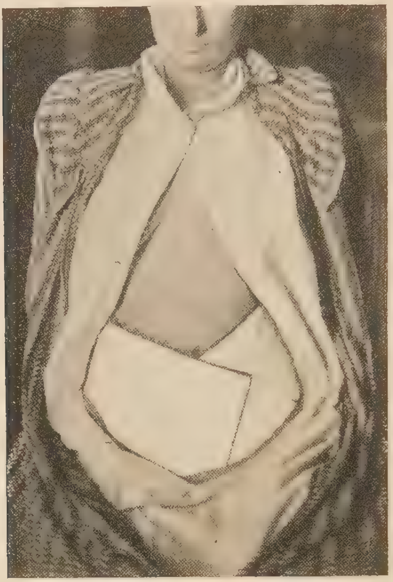

Fig. 135.—Rose's belt: Complete.

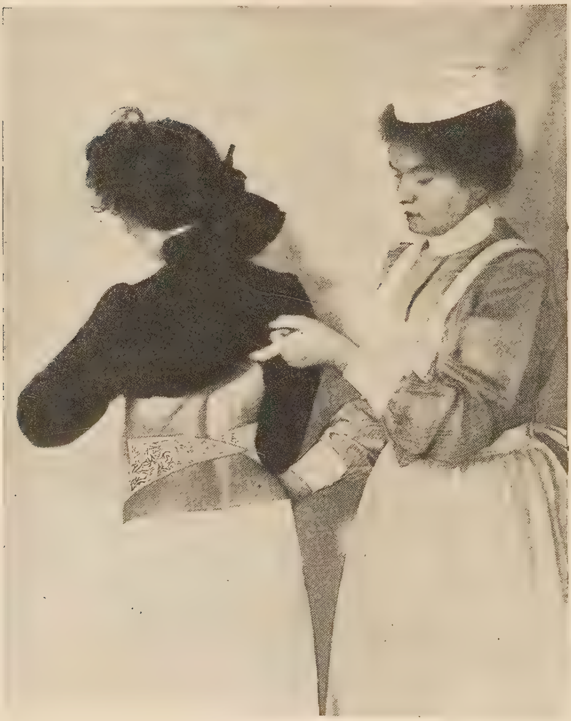

Fig. 136.—Dorsal view: Under plaster with overlapping ends (Rose and Kemp).

*I* is applied to the abdomen, and the lateral pieces, *II*, *III*, overlap in front and are applied to the under plaster. These serve to draw up the abdomen.

To avoid irritation of the umbilicus, I cut a V out of the upper border of the under plaster or invert a small portion of it. The sharp angle below should be cut off to avoid interference with the pubic hair. The curved portions of the lateral wings should look upward and somewhat inward and adhere to the lower ribs. The sharp angles of the lateral wings at the symphysis may also be cut off to avoid the hair.

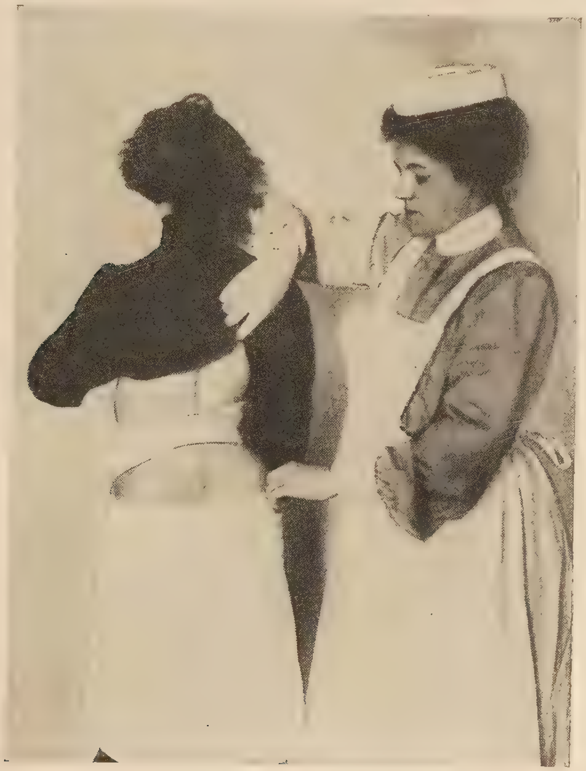

Fig. 137.—Dorsal view: Plaster dressing complete (Rose and Kemp).

Hair, if present on the abdomen, is shaved and the surface cleaned with ether or chloroform.

The plaster is applied with the patient in the dorsal position, and preferably with hips slightly elevated.

In the illustrations (Figs. 133–135) are shown the three stages of application of the belt, the under plaster, one wing applied, and the complete contrivance. In Figs. 136 and 137 are shown the rear view during application.

The plaster should remain on for four to six weeks, depending on the season of the year, irritation (which is rare), or its loosening. It should then be removed, a full bath given, talcum dusted on, and twenty-four hours later a new belt applied.

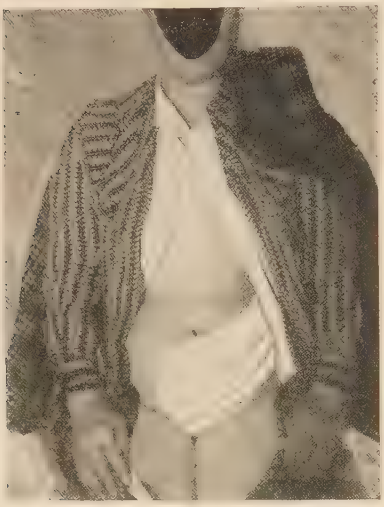

Fig. 138.—Application of narrow strips of adhesive plaster: First step.

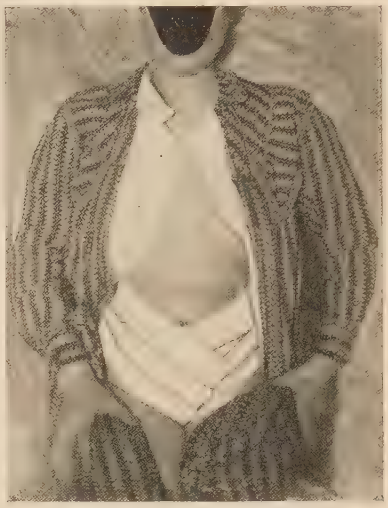

Fig. 139.—Application of plaster: Second step.

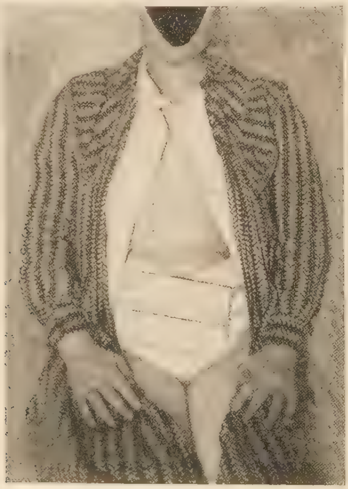

Fig. 140.—Application completed.

Oil of wintergreen, applied to adhesive plaster, aids its easy and painless removal. One can also apply to the plaster a 10 per cent.[1] wintergreen oil ointment.

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[1] Beardsley, Jour. Amer. Med. Assoc., Jan. 28, 1911.

One patient sent me by Wm. H. Thomson, a severe case of splanchnoptosis, wore the belt fourteen months, gained 40 pounds in weight, and was completely cured.

The device gives brilliant results. The method of support by a pad for the special organ is unscientific.

Only on occasions, when the material for Rose's belt was not at hand, have I applied a method with narrow strips of plaster, as depicted in Figs. 138–140. They overlap at the linea alba in front and at the spine behind. As the final procedure, two transverse strips are applied in front.

**Rosewater Adhesive Belt.**—A strip of zinc oxid plaster of sufficient length and 2 to 3 inches wide is fastened to the abdomen just above the pubes. This is drawn upon upward and fastened above to the lower end of the sternum. Diagonal strips crossing the lower end of the vertical strip, overlapping behind at the spine, are then applied (Figs. 141 and 142).

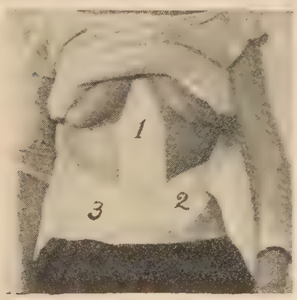

Fig. 141.—Front view: Step one.

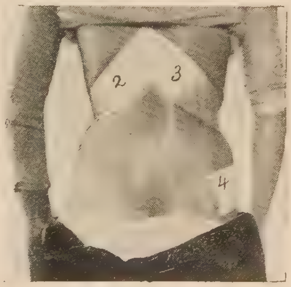

Fig. 142.—Rear view.

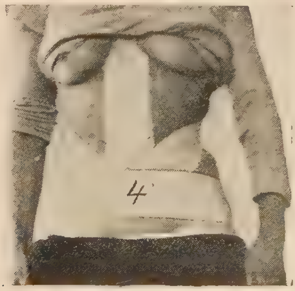

Fig. 143.—Front view. Belt complete.

Figs. 141–143.—Rosewater adhesive plaster belt (Rose and Kemp).

A horizontal strip is fastened to one hip and stretched across the pubes to the other hip, overlapping the ends of the other plaster and acting as an additional girdle (Fig. 143).

Plaster strapping affords continuous support during treatment which other methods do not. Numerous modifications, with and

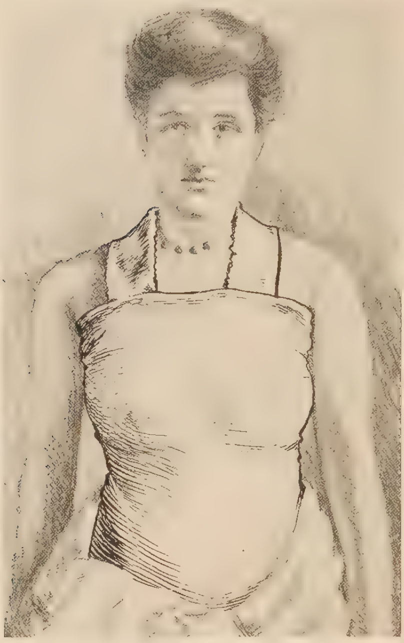

Fig. 144.—Kilmer's belt stockinet band: Step one.

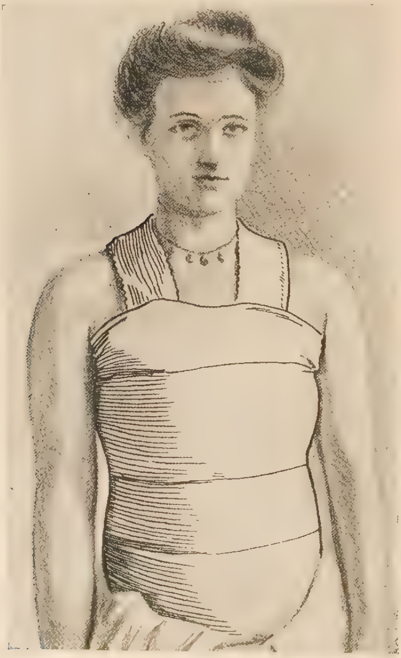

Fig. 145.—Kilmer's belt (complete): Elastic webbing, front.

without eyelet holes for lacing, have been devised, but those described are the most practical.

**Kilmer's Abdominal Belt.**—An ingenious belt was devised some years ago by T. W. Kilmer for the relief of vomiting in pertussis. The original belt consisted of a stockinet band applied as in Fig. 144. Around this was wound a strip of silk elastic webbing, which could be pinned or sewed on, as in Figs. 145 and 146.

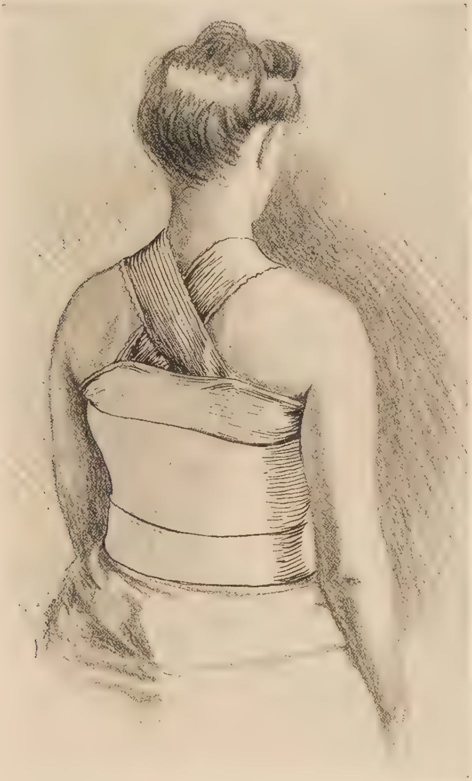

Fig. 146.—Kilmer's belt (complete): Elastic webbing, rear.

The apparatus extends from just above the hips and symphysis well up on the thorax. The relief of vomiting and cough is quite remarkable in pertussis.

Recently, Kilmer reports a simplified belt made of linen, with strips of elastic webbing inserted on either side. It laces up the back (Figs. 147 and 148).

The belt should measure slightly less (2 to 3 inches) than the circumference at the navel. The degree of constriction should be determined in every case. This belt is valuable for the prevention

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[1] Archives of Pediatrics, February, 1907.

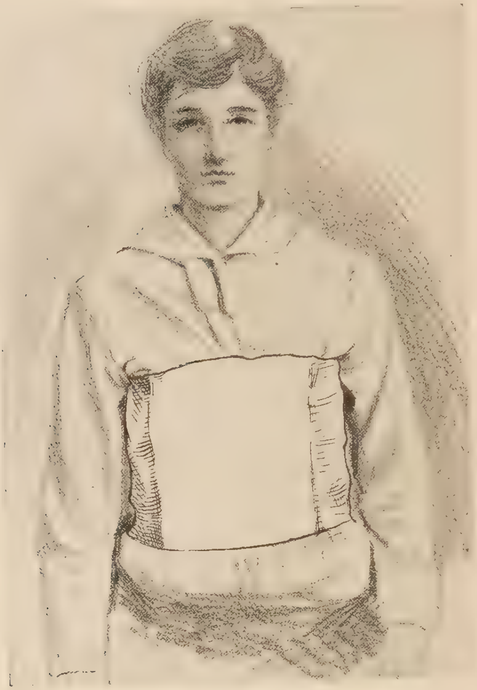

Fig. 147.—Improved belt (front).

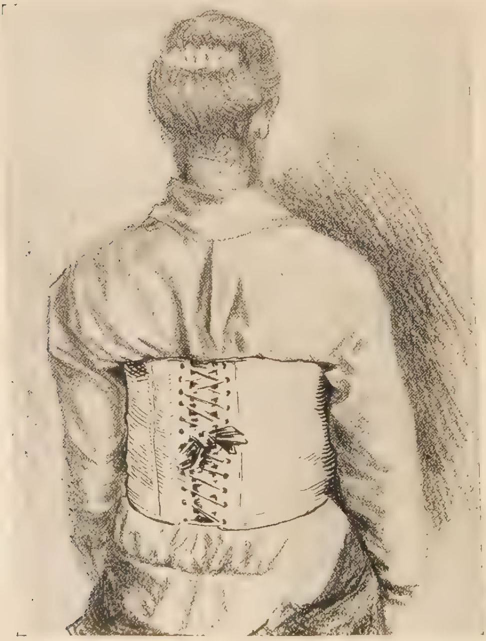

Fig. 148.—Improved belt (rear).

of seasickness, for nervous vomiting, and as an abdominal support. The cost is slight.

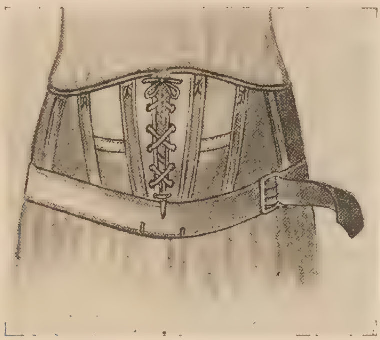

Fig. 149.—Teufel's abdominal supporter.

**Silk Elastic Belts.**—Various types of silk elastic belts are employed as pictured in Figs. 149–151. Storm's abdominal supporter is also excellent.

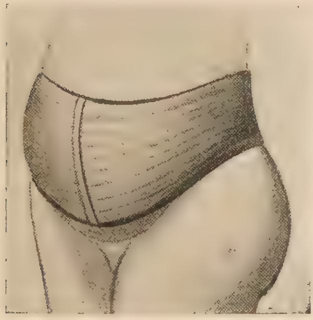

Fig. 150.—Abdominal supporter.

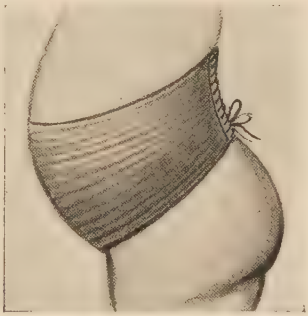

Fig. 151.—Abdominal supporter.

**Special Corsets.**—A valuable support of this type is that of E. Gallant, which is depicted under Gastroptosis. The La Grecque surgical corset is also useful, and is illustrated in the same chapter. The latter is frequently employed by the writer.

## CHAPTER X

# CATARRH OF THE STOMACH

## ACUTE AND CHRONIC GASTRITIS

### ACUTE GASTRITIS

ACUTE gastritis may be defined as an acute inflammation of the gastric mucous membrane with resulting disturbances of digestion. It is of different degrees of severity, being limited to the superficial layer of the mucous membrane, or it may extend to the glandular parenchyma or involve the interstitial tissues.

It is subdivided into simple acute gastritis, toxic gastritis, and phlegmonous gastritis.

### SIMPLE ACUTE GASTRITIS

(*Synonyms.*—Acute Gastric Catarrh; Acute Gastric Dyspepsia.)

**Etiology.**—Simple acute gastric catarrh is one of the most frequent diseases met with by physicians. It occurs in all classes of society and at all ages. It may be primary or secondary to another disease. One of its frequent causes is some irritant, mechanic, chemic, or thermal; thus, errors in diet, or too large a quantity of food that has been imperfectly masticated or rapidly bolted; too hot or too cold food or drink; too highly spiced or fermented foods; rancid butter; unripe or spoiled fruit; spoiled food or drink, or overindulgence in alcohol.

Fermentation or putrefaction are most apt to occur in food during the summer, and these factors probably account for the epidemics occurring at that season, though infection has been suggested. Such cases generally occur as gastro-enteritis.

Bacterial infection of food may be a cause, as the colon bacillus in milk or meat, or sausage poisoning.

Primary mycosis of the stomach, the favus fungus; schizomycetes; parasites, as the larvæ of flies (myiasis); ascarides, oxyuris and tænia, by entering the stomach, and abdominal burns are rare causes. Acute catarrhal or suppurative conditions of the nose and throat may produce acute gastritis from the ingestion of discharges. I have recently seen one such case, and the patient, suffering from acute gastritis, cured by treatment of the source.

Some persons have a predisposition to a " weak stomach," and this condition seems almost to be hereditary. Others have been trained to such a simple diet, as the children of dyspeptics, that the stomach cannot perform its normal amount of work and readily becomes irritated. In old persons, invalids, or anemic women the organ is readily affected.

Acute gastritis may be secondary to the acute infectious diseases, such as measles, typhoid, variola, pneumonia, etc., or as a sequel of acute nephritis. I have seen an attack follow prolonged anesthesia.

A diphtheritic or membranous gastritis has been met with in diphtheria, anthrax, or as a secondary process in typhoid, typhus, pneumonia, etc. It cannot be diagnosed unless the membranes are vomited.

**Morbid Anatomy.**—Acute gastritis is characterized by an acute inflammation of the superficial layers of the mucosa, with an increased secretion of mucus and a desquamation of the epithelial cells.

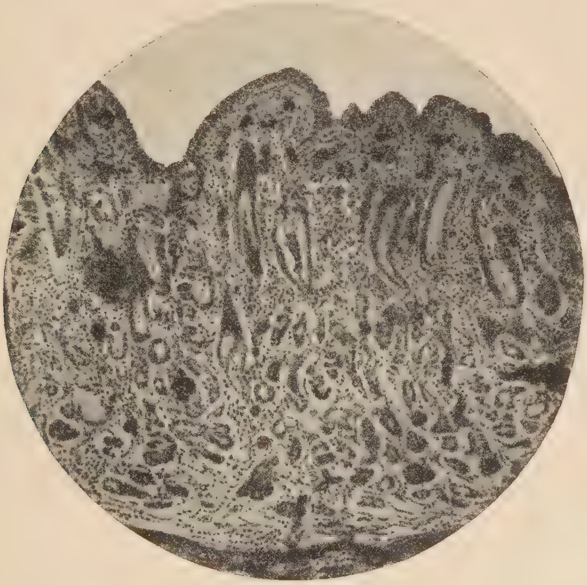

Fig. 152.—Acute gastritis: Round-celled infiltration in the interglandular structure. Dark clumps represent hemorrhagic areas. (From Bassler's "Diseases of the Stomach and Upper Alimentary Tract." Copyright, 1910, by F. A. Davis Company.)

The mucous membrane is reddened and swollen, less gastric juice is secreted, and mucus covers the surface. The swelling is diffuse or in circumscribed areas. There are sometimes slight hemorrhages and small erosions or sacculations of the mucous membrane. The submucosa may be edematous. The pyloric end is more frequently affected. Gastric secretion is weakly acid, neutral, or even alkaline and diminished in quantity. Beaumont, from his observations on St. Martin, has given an excellent description.

**Microscopic.**—The superficial epithelial layer is partially loosened or in a condition of cloudy swelling. The parietal and principal cells cannot be distinguished apart; they are granular and in a condition of cloudy swelling and fatty degeneration, and are shrunken.

The capillaries are dilated and round cells are found in the interglandular tissue, between the epithelial cells and on the surface (Fig. 152). Karyokinesis may be present.

Bacteria are commonly found present, of which the most frequent types are the Bacillus lactis aërogenes, Bacillus coli communis, proteus vulgaris, oïdium albicans, and streptococci.

**Symptoms.**—These vary according to the severity of the attack. There are loss of appetite, discomfort, fulness or pressure in the region of the stomach, belching of gas which may taste sour, and occasional nausea. In some cases there are no rise of temperature and no vomiting, and the symptoms pass off in a day or two; the bowels are costive or diarrhea is present.

In more severe cases there are pains in the gastric region, headache, nausea, vomiting (prolonged and excessive), first of food, then chiefly of mucus, at times streaked with blood, and frequently bilious vomiting. There may be considerable prostration. Often there is an acid taste in the mouth. There is generally a temperature, sometimes rising to 102° to 104° F., and at times chills, and the tongue is usually coated and swollen. The pulse is frequently rapid and feeble.

In the cases due to ingestion of spoiled food, etc., auto-intoxication undoubtedly results and aggravates the symptoms.

Vomiting usually follows the introduction of the irritant, but is sometimes delayed for some hours, and food is found that was ingested twelve or fifteen hours before, a condition of acute motor insufficiency. Constipation or diarrhea is present. The early vomitus often has a disagreeable odor and contains food remnants. The reaction is slightly acid and free hydrochloric acid is often absent. Lactic and other organic acids are at times present. Occasionally a *duodenitis with jaundice is associated.* Herpes labialis is quite frequent.

**Physical Examination.**—The region of the stomach is usually distended and sensitive, and may be tender on pressure.

**Urine** is scanty, dark in color, of high specific gravity, urates are marked, and occasionally indican and albumin are present.

**Duration.**—This is usually short, from two to three days, though at times prolonged to a week.

**Diagnosis.**—Some of the infectious diseases, notably scarlatina, begin like the febrile form of acute gastritis, and one should always be on the watch for such an occurrence.

In rare cases the symptoms are intensely severe, headache and even delirium being so marked as to have been mistaken for meningitis. In the latter case, Kernig's sign can be elicited and lumbar puncture is an aid to diagnosis.

In biliary colic, with acute vomiting, the pain radiates to the right side or right shoulder, and pain over the gall-bladder is present.

In cholecystitis, with or without calculi, with little or no pain and no jaundice, but vomiting, the diagnosis is more difficult. The previous history, tenderness over the gall-bladder, and the presence of leukocytosis, especially the increase in the polynuclears, are significant. Hyperchlorhydria is frequently associated. The presence of Head's gall-bladder zone of cutaneous algesia aids diagnosis.

With peritonitis, we have muscular rigidity, leukocytosis, increased polynuclears, marked abdominal tenderness, and distention.

With nervous gastralgia, the material vomited is usually very acid (hyperchlorhydria), no mucus, and there is the nervous history.

With typhoid fever, we have the gradual rise of temperature, increasing daily, the splenic enlargement, frequently the eruption, often bronchitis, Ehrlich's diazo and the Widal reactions; while with acute gastritis the rise of temperature is sudden and the fall equally sudden, and there are the absence of splenic enlargement and other symptoms.

When jaundice is associated with acute gastritis, the duodenum has evidently become involved. The gastric crises of locomotor ataxia have been mistaken for acute gastritis, but the absence of knee-jerks, the Romberg symptom, and Argyll-Robertson pupil are diagnostic of tabes.

**Prognosis.**—This is favorable, except in very old people and invalids.

**Treatment.**—*Prophylaxis.*—In patients subject to attacks of acute gastritis, excess in eating, rich food, lobster, food and drink that are too hot or cold, or any articles for which they have an idiosyncrasy should be forbidden. Candy and cake should not be allowed. Unripe and dirty fruit should be avoided. With infants, care should be taken as to the storage of milk and its preparation.

If the acute gastritis be due to ingestion of improper food, there are two principles to follow: Clear out the gastro-intestinal tract and give rest to the stomach. If there are other factors, the first consideration does not apply. In all cases *rest in bed should be enjoined.*

In the *mild cases*, with *nausea* but *no vomiting*, castor oil, 1 to 1½ ounces (32.0–48.0); or, if there is doubt of this being retained, then calomel, 2 to 5 grains (0.125–0.3), or blue mass, 5 grains (0.3), followed in twelve hours by a saline, such as a wineglass of Apenta, or citrate of magnesia (wineglass), or magnesium sulphate, 1 to 2 drams (4.0–8.0), should be administered. Laxol, a preparation of castor oil without the disagreeable taste, is often well borne in the same dosage as castor oil.

If much nausea, calomel, $\frac{1}{10}$ grain (0.006), with sodium bicarbonate, ½ grain (0.0325), every hour for eight or ten doses, followed by a saline. Other combinations are given on page 214.

Children should receive proportionately small doses of cathartics.

For eructation of acid fluid (pyrosis), bismuth subnitrate, 3 grains (0.2), with sodium bicarbonate, 3 grains (0.2), every hour or two;

or magnesia usta, ½ ounce (16.0), with sodium bicarbonate, ½ ounce (16.0), as much as covers the point of a kinfe, every three hours.

For nausea or vomiting, oxalate of cerium, 1 grain (0.065), every hour for several doses, or bismuth subnitrate, 2 to 4 grains (0.125–0.25), alone, or with sodium bicarbonate, same dose; or gelatin (1 per cent. solution), dose 1 dram (4.0), every hour, given cold; or milk, 8 ounces (250.0 c.c.), with oxalate cerium, 10 grains (0.6) and sodium bicarbonate, 10 grains (0.6), packed in ice; dose, 1 dram (4.0) every hour; or Fowler's solution of arsenic, 1 minim (0.06), every hour for four doses, are of value. This last is very efficacious.

Teaspoonful doses of hot water or, occasionally, a piece of cracked ice, or white of raw egg beaten up and given cold in 1-dram doses (4.0), every half-hour to an hour, are useful.

*Cocain, carbolic acid, or creosote I strongly deprecate.* Heat should be applied.

*Application of Heat.*—A hot-water bag, one-third full to avoid weight; a Japanese hot box, a light hot salt-bag, a thin tin pie-plate heated in the oven and covered with flannel; or moist heat by means of a flaxseed poultice, hot mashed potato, or bread poultice in country practice; mustard and flour poultice (equal parts); or black or red pepper poultice—1 dram (4.0) to 1 pint (500 c.c.) of boiling water—and wring out flannel in the same and apply, covering with oiled silk.

If the discomfort is marked, then give warm salt water, ½ to 1 dram (2.0–4.0) of salt to 8 ounces (250 c.c.) of water, and tickle the fauces to promote vomiting, or perform lavage.

Small doses of hot water may be given to relieve thirst and *food should be avoided for twenty-four hours.*

If small quantities of nourishment be given, I have found 1 ounce (32.0) doses every hour of a 5 per cent. gelatin solution (in a glass packed in ice) of special value, or small quantities of milk and lime-water (equal parts), or peptonized milk or koumiss.

In *severer cases,* the stomach should be emptied, preferably by lavage, 1 to 2 ounces (32.0–64.0) of Phillips' milk of magnesia to 2 quarts (liters) of warm water being excellent for this purpose. Plain warm water or normal salt solution may be employed.

I frequently administer calomel, 3 to 5 grains (0.2–0.3), and sodium bicarbonate, 5 grains (0.3), in a little water, through the stomach-tube after lavage before withdrawal of the tube. This is generally retained, with resulting thorough clearing of the bowels. Saline solution should not be employed for lavage if calomel is thus given.

Later an enema of a saturated solution—2 to 3 ounces (64.0–96.0)—of magnesium sulphate, or a recurrent enteroclysis with normal saline solution at 110° F. should be administered (several quarts—liters), or a soapsuds enema, 1 quart (liter), containing olive oil, 6 ounces (200 c.c.), may be substituted. Even if the calomel be omitted, it is of great importance to move the bowels. If the case is not due to ingestion of irritating food, then bismuth subnitrate, oxalate of cerium, a few doses of $\frac{1}{10}$ grain (0.006) of calomel every hour, sodium bicarbonate, etc., may be tried for a brief period; and if these fail, lavage should be performed. Gelatin solution or white of egg often are of value in such cases.

With bilious vomiting, lavage should be employed at once, as there is practically reversed peristalsis with an open pylorus, and the continuous accumulation of bile in the inflamed stomach keeps up the vomiting. Enteroclysis should be used after lavage (within one hour) to promote normal peristalsis.

It may be necessary to wash the stomach several times, but it is the best method to check vomiting.

In the severe cases, due to ingestion of improper food, it is my opinion that calomel should be given directly after lavage, even though some of it be vomited, as auto-intoxication is a factor.

In addition to the milk of magnesia, if the vomitus is very foul, I add 10 grains (0.6) resorcin to the fluid for lavage and wash with the patient both in the erect and lying-down position. I do not care for apomorphin, $\frac{1}{10}$ grain (0.006), ipecac, or tartar emetic to empty the stomach.

If there is considerable prostration, enemata of hot normal salt solution (115°–120° F.) and strychnin by hypodermic—$\frac{1}{60}$ to $\frac{1}{30}$ grain (0.00108–0.002)—may be necessary, or even 5 grains (0.3) camphor in 20 minims almond oil by hypodermic.

In rare cases, when there has been much vomiting and the patient is exhausted, codein, $\frac{1}{8}$ to $\frac{1}{4}$ grain (0.008–0.016), by hypodermic, or the same amount of morphin may be used. I sometimes employ a single dose after lavage to quiet the patient. A suppository—1 grain (0.064) opium, or $\frac{1}{4}$ grain (0.016) morphin with $\frac{1}{3}$ grain (0.021) extract belladonna—may be substituted.

*Diet.*—Entire abstinence from food during the first twenty-four hours or longer is preferable; nutritive enemata may be given and injections (rectal) of hot saline solution to relieve thirst.

For the latter, 1-dram (4.0) doses of hot water by mouth, or of a cold 1 to 2 per cent. gelatin solution are preferable to cracked ice. The gelatin seems to have an excellent effect. Proctoclysis is also excellent for the same purpose.

Later, small doses of cold gelatin (5 per cent. solution) or milk and lime-water (equal parts) can be given, or milk, 8 ounces (250 c.c.), with sodium bicarbonate, 10 grains (0.6), and cerium oxalate, 10 grains (0.6), in $\frac{1}{2}$- to 1-ounce (16.0–32.0) doses, given cold, every two or three hours.

White of egg beaten up and given cold; barley-water alone or with milk (equal parts); rice gruel, very thin, made from rice flour (Park and Tilford) with milk may be added.

Later, add eggs (soft boiled), scraped raw beef, pigeon (boiled), calves' brains, zwieback, broths, soups, bouillon, boiled chicken, and gradually increase to full diet.

If after the acute attack has subsided the patient suffer from a feeling of pressure and discomfort, dilute hydrochloric acid may be given to aid digestion.

℞. Acid. hydrochlor. dilut. . . . . . . . . . . . . . . . . . . . . . . . ʒiiss (10.0);
Aq. destil. . . . . . . . . . . . . . . . . . . . . . . . . . . .q. s. ad. ℥ij (60.0).
Sig.—ʒj to ij (4.0–8.0), in water, t. i. d.

The same, combined with small doses of tincture of nux vomica,[1] 5 minims (0.3) in each dose, or with compound tincture of cinchona, 10 minims (0.6) in each dose, may be given.

These remedies should be administered one-half hour after meals or the same time before.

### TOXIC GASTRITIS

**Etiology.**—This most intense form of inflammation of the stomach is caused by the swallowing of concentrated mineral acids or strong alkalis, or by poisons, such as phosphorus or arsenic. Among such acids are nitric, sulphuric, hydrochloric, oxalic, and carbolic; the caustic alkalis, as caustic potash, caustic soda, soap lees, and strong ammonia; alcohol, phosphorus, arsenic, potassium cyanid, corrosive sublimate, and potassium chlorate. The effects are more severe on the empty stomach. Croton oil may also produce this condition.

**Anatomy.**—The acids and alkalis destroy the parts they come in contact with, causing various degrees of sloughing of the mucous membrane. They may penetrate the submucosa or the entire stomach wall and produce perforative peritonitis.

Alcohol, phosphorus, or arsenic cause an acute inflammation of severe type, the mucous membrane becoming swollen and superficially necrotic, with hemorrhagic spots in the submucosa, and there is fatty degeneration of the epithelia of the glandular tubuli.

**Symptoms.**—There is intense pain in the gastric region, violent and burning in character, and increased on pressure; and frequently pain in the pharynx and esophagus (along the sternum). There are salivation, difficulty in swallowing, and usually vomiting, constant and repeated, which fails to relieve the pain. This is generally immediate, though not always so. There may be food remnants in the vomit, mucus, streaks of blood, and even shreds of mucous membrane. The abdomen is tender, at times distended, though occasionally contracted. Symptoms of collapse often appear, the face is pale and anxious, the skin pale and extremely cold, pulse rapid and feeble, respiration rapid and shallow. There are restlessness and sometimes convulsions.

Albumin and blood often are present in the urine and petechiæ under the skin. The writer has recently seen a fatal case of bichlorid poisoning with marked hemorrhagic tendency. Peritonitis, shock, or respiratory or cardiac failure may cause the fatal issue.

Some cases are more protracted. There may be jaundice or hematuria, or intestinal ulceration, or degeneration of the liver or kidneys. Phosphorus-poisoning may produce hemorrhagic jaundice and symptoms simulating acute yellow atrophy of the liver.

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[1] Oxyntin capsules with nux vomica, one to two capsules t. i. d., a. c. or p. c., are an excellent substitute.

Stricture of the esophagus, pylorus, or cardiac orifice of the stomach may result from the damaged mucous membrane, with corresponding symptoms, such as dilatation of the stomach, etc. In rare cases atrophy of the mucous membrane (achylia gastrica) follows, or hour-glass stomach, or perigastric adhesions.

**Diagnosis** is usually easy. The sudden appearance of violent gastric symptoms in a perfectly healthy subject should excite suspicion.

Inspection of the lips, mouth, and tongue will show the effect of corrosive poison, if such have been taken, and examination of the vomitus and odor of the breath may afford information. Inspection, if possible, of the receptacle from which the substance was taken, and examination of the latter, if any remain, are important. The patient often gives a clear history.

**Prognosis.**—It is best to give a guarded prognosis even in apparently favorable cases.

**Treatment.**—There are certain cardinal rules to follow in the treatment of toxic gastritis: first, administer fluid to dilute the poison, and at the same time give an antidote; empty the stomach as rapidly as possible, preferably by lavage; administer demulcents; stimulate the patient and give a cathartic to clear the poison from the bowels.

Though some advise against lavage in poisoning from acids and alkalis for fear of perforating the stomach, there is far greater danger of perforation by leaving the poison in the organ, with the additional risk of cardiac or respiratory failure, or subsequent damage to the intestines or other viscera.

Lavage by siphonage of the stomach is indicated *in all cases of poisoning*. Warm water at about 101° F. should be employed, and the organ washed until thoroughly clean. Any bland fluid, such as tea, coffee, soup, water, or milk, can be used in an emergency to dilute the poison and wash the stomach.

A gastric siphon can be improvised from a fountain syringe by removing the tip and clip and rounding the edges of the extremity; or from a kitchen funnel and rubber tube of small caliber.

If lavage be impossible, then give a pint of lukewarm water with 2 drams (8.0) of mustard dissolved therein, or warm salt water, and tickle the fauces with the finger or a feather.

Apomorphin, $\frac{1}{10}$ grain (0.006), by hypodermic; zinc sulphate, 30 grains (2.0), in 4 ounces (30.0) of water; copper sulphate, 5 grains (0.3), providing these latter were not the poisons ingested; or syrup of ipecac, 1 to 3 drams (4.0–12.0), can be employed.

Among useful *demulcents* are whites of raw eggs, milk, olive oil, barley-water, flour boiled with water, and gum-arabic water. Fats and oils *should be avoided in phosphorus-poisoning*. The subsequent treatment is of acute gastritis, enteritis, and of special symptoms.

**Antidotes.**—*For Acid Poisoning.*—Alkalis, such as calcined magnesia; powdered chalk in water; sodium carbonate (washing soda), dilute; potassium carbonate; sodium or magnesium sulphate, ½ ounce (16.0), in water; Carlsbad salts; soapsuds in water. Soda bicarbonate, 1 ounce (16.0), or, if nothing else is at hand, plaster scraped from the wall, dissolved in water, 8 ounces (250 c.c.), can be employed.

*For Carbolic Acid.*—Alcohol (95 per cent.), 1 to 3 ounces (32.0–96.0); raw whisky or brandy, or liquor with a large percentage of alcohol. The alkalis can be used subsequently or alone if alcohol or liquors are not obtainable.

*For Caustic Alkalis.*—Dilute acids, such as dilute vinegar or lemon juice; tartaric or citric acid.

*For Tartar Emetic and Antimony and Its Compounds.*—Tannic acid or strong tea.

*For Arsenic and Its Compounds.*—Tincture of perchlorid of iron, 1½ ounces (48.0) in a wineglass of water, add ½ ounce (16.0) sodium carbonate (washing soda) in half-tumbler of water, mix, and administer. This renders insoluble about 5 grains (0.3) of arsenic. Repeat dose, or give dialyzed iron, tablespoonful doses.

*For Copper Salts.*—Potassium ferrocyanid, 1 dram (4.0) to 4 ounces (125 c.c.) of water, forms insoluble copper cyanid.

*For Iodin, Iodids, and Iodoform.*—Starch solution in cold water; sodium bicarbonate; lead acetate, 2 drams (8.0) in 4 ounces (125 c.c.) of water.

*For Lead and Its Salts.*—Magnesium or sodium sulphate, 1 ounce or more, or dilute sulphuric acid, 30 minims (2.0), in water.

*For Mercury and Its Salts (Corrosive Sublimate, etc.).*—White of raw egg; milk; form albuminate.

*For Silver Nitrate.*—Salt solution, teaspoon of salt in water, 4 ounces (125.0).

*For Zinc Salts.*—Sodium or potassium carbonate; tannic acid; tea; white of egg; milk.

*For Phosphorus (Rat Paste, Matches).*—Copper sulphate, 3 to 5 grains (0.2–0.3), in 4 ounces (125 c.c.) of water, a number of doses; forms insoluble phosphid of copper and acts as an emetic; old French turpentine. Avoid oils, fats, milk, and yolks of eggs. Avoid American or German turpentine.

*For Alcohol.*—Ammonium carbonate, 3 grains (0.2), in water.

The stomach should receive rest after immediate treatment. Pain may be relieved by local heat, or codein or morphin, ⅛ to ¼ grain (0.008–0.016), hypodermically. Large doses of bismuth subnitrate may be given if the bowels continue irritable. Retention or suppression of urine must be watched for. In the latter case, enteroclysis, with hot normal salt solution at 115° to 120° F. Stenosis of the esophagus may be dilated if possible; if stenosis of the pylorus, appropriate treatment as for gastric dilatation, and in most cases subsequent operation. If atrophy of the gastric mucosa result, the treatment is for achylia gastrica. For perforative peritonitis operation is indicated.

## PHLEGMONOUS GASTRITIS

(*Synonyms.*—Suppurative Inflammation of the Stomach; Gastritis Phlegmonosa; Abscess of the Stomach.)

This is a rare disease and usually runs an acute course, though occasionally subacute. The process begins in the submucosa and sometimes extends to the muscular coat, and at times to the mucous or serous coats. It is more frequent in men. It is primary or idiopathic, due to some micro-organism, especially the streptococcus, probably entering through some solution of continuity in the mucous membrane; or secondary (metastatic), due to pyemia, puerperal infection, or the exanthemata.

Errors in diet, alcoholic excess, trauma, etc., have been given as causes, but probably only are contributory by depressing the system. Traumatism might cause damage to the mucosa and render infection more easy.

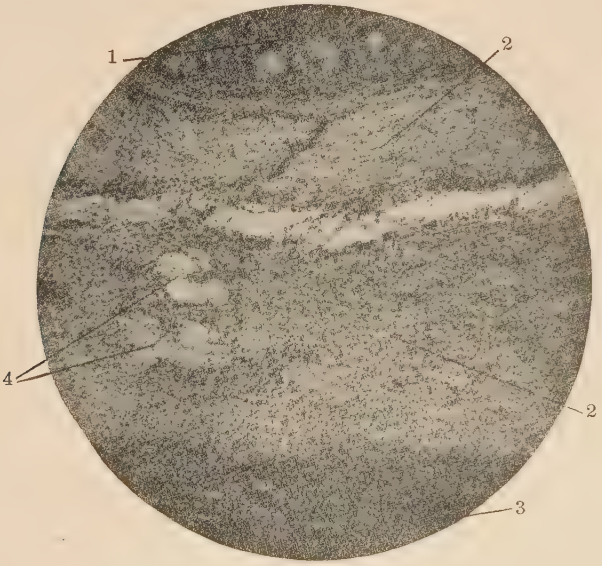

Fig. 153.—Phlegmonous gastritis: 1, Gastric mucosa infiltrated with pus-cells in the interglandular tissue; 2, submucosa thickened and infiltrated; 3, purulent collection oozing out on section; 4, fat tissue. (From Bassler's "Diseases of the Stomach and Upper Alimentary Tract." Copyright, 1910, by F. A. Davis Company.)

*Age.*—Cases have been reported from ten years of age to eighty-five. It is probably most frequent in early adult and middle life.

There are two forms met with: a diffuse purulent infiltration and a circumscribed abscess.

**Morbid Anatomy.**—In the diffuse type a large area of the submucosa is frequently involved. It is thickened, infiltrated with pus, and multiple small abscesses are often present (Fig. 153). The pyloric end is more frequently attacked. The muscular wall is often involved or the pus may burrow through to the peritoneum. The mucosa is usually also affected and is swollen, and there is granular degeneration of the gland cells. Perforation of the mucosa may occur.

In the abscess type there is generally a single circumscribed abscess of variable size, starting in the submucosa and involving the muscular layer. The mucosa and serosa are often involved. It may terminate favorably by perforating into the stomach (a rare event) or perforate into the peritoneal cavity.

**Symptoms.**—The patient may have for a brief period a few dyspeptic symptoms, such as loss of appetite, thirst, and some burning in the stomach, but these are usually absent.

The attack is generally of an acute fulminating type: Severe pain or burning in the gastric region, a rapid rise of temperature to 103° to 105° F., with slight intermissions; frequently chills; often vomiting of mucus, bile, and food remnants, but no pus, unless the abscess breaks into the stomach, which is a rare event. The area over the stomach is very sensitive to pressure and there is some tympanites. There is constipation or, more usually, diarrhea. The pulse is rapid and feeble, occasionally there is jaundice.

The patient presents all the aspects of a severe infection, with delirium and coma preceding death. The blood examination shows *leukocytosis* with increase in the *polynuclears*. There is *muscular rigidity* in the *upper quadrant* of the abdomen, of the recti muscles, due to peritoneal irritation, even before perforation occurs. This is a valuable sign. With general peritonitis we have the usual symptoms —pain, distention, general tenderness on pressure, etc.

With a circumscribed abscess the tenderness is more localized, the symptoms not so severe, and the duration longer. Percussion and palpation may locate the process if localized.

**Duration.**—Three or four days, rarely one to two weeks.

**Diagnosis.**—The temperature, chills, fulminating character, leukocytosis, and early recognition of muscular rigidity—all point to an acute suppurative process. Abscess of the liver and subphrenic abscess are not of such acute type. Acute cholecystitis and acute pancreatitis are more apt to be confounded with abscess of the stomach.

With acute pancreatitis the temperature at first may be low, tympanites is earlier and more marked, and there are circumscribed tenderness in the course of the pancreas and tender spots throughout the abdomen (Fitz). Constipation is usual; also the symptoms may resemble intestinal obstruction. Abscess of the pancreas is slower in its course and there is a longer history.

Acute cholecystitis is a much more frequent condition than phlegmonous gastritis. The gall-bladder can at times be palpated, is tender, and we have the previous history.

**Treatment.**—Laparotomy is advised, both to settle the diagnosis and afford relief if possible. Should this not be consented to, then the ice-bag, rectal feeding, enteroclysis to relieve tympanites, and opiates are indicated.

Bovée[1] reports a case with operation and recovery.

## CHRONIC GASTRITIS

(*Synonyms.*—Chronic Gastric Catarrh; Chronic Dyspepsia.)

**Definition.**—A chronic inflammation of the mucous membrane of the stomach, with the production of mucus and changes in the gastric juice, causing disturbances in the act of digestion.

**Etiology.**—This disease is more frequent in men than in women. It may follow the acute type, especially after recurrences of this condition. The same irritating agents that cause acute gastritis can produce the chronic type when acting for a long period of time; notably, fast eating with imperfect mastication, overloading the stomach, indigestible food, highly spiced dishes, cold drinks in excess, overrich food, excessive use of tea or coffee, and overindulgence in alcohol (the so-called "drunkards' catarrh"); excessive use of tobacco, especially in tobacco-chewers and those who indulge in dry smoking (chewing the cigar-butt), an unhealthy condition of the mouth or teeth, and swallowing the products of decomposition. Habitual use of drugs may be a cause.

Discharges from the nose or ear through the Eustachian tube, which are then swallowed, have been factors in several cases that I have observed. Thrush may cause mold in the stomach. Chronic gastritis may be secondary to the acute infectious diseases, such as typhoid fever. It is frequently associated with cancer of the stomach and is often present in the atonic type of dilatation of the stomach.

It may be secondary to cirrhosis of the liver, pulmonary or cardiac disease, and chronic nephritis and syphilis. Among constitutional causes are gout, diabetes, leukemia, and severe anemia.

**Classification.**—Chronic catarrhal gastritis may be classified as follows:

1. *Acid gastritis* (gastritis hyperpeptica, hypersthenic gastritis), or acid catarrh of the stomach, first described by Boas.

This is considered on the borderline and to be the initial stage of chronic gastritis. There is a slight increase in the hydrochloric acid, and Boas believes this occasionally occurs in the early stages of chronic gastritis. There is also the presence of mucus.

2. *Chronic catarrhal gastritis*, under which is described mucous gastritis (Ewald), which is merely a severe type with great secretion of mucus.

We must remember that occasionally from a long-continued chronic gastritis, other conditions may arise.

(*a*) There may be a great thickening of the mucosa (hyperplasia), so as to produce thick folds, the so-called etât mamelonné, and this, combined with swelling of the mucosa, causes benign stenosis of the pylorus; or sometimes wart-like excrescences (gastritis polyposa) develop, which, if situated at the pylorus, can produce partial obstruction—a benign stenosis, with dilatation of the stomach; or a proliferation of interstitial tissue occurs and muscular hypertrophy, with resulting hypertrophic stenosis of the pylorus.

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[1] Amer. Jour. Med. Sci., May, 1908.

Chronic gastritis may, therefore, produce and have associated with it benign pyloric stenosis and dilatation of the stomach.

(*b*) On the other hand, a degeneration or atrophy of the muscular fibers due to hyperplasia of the connective tissues may cause atony, and atonic dilatation of the stomach may result, and these conditions will improve with the improvement of the gastritis.

They only occur in the more advanced cases. In the patients whom we are called upon to treat, the stomach is *usually of normal size,* non-atonic, and with normal or slightly diminished motor functions.

3. *Atrophic gastritis* (Anadenia ventriculi, Ewald), an atrophy of the mucous lining of the stomach, can result from chronic gastritis. Of these, there are two forms:

(*a*) Phthisis ventriculi, a thinning of the coats of the organ, which may remain of normal size or be increased in size.

(*b*) Cirrhosis, or sclerosis ventriculi, an enormous thickening of the muscular coat and a great reduction in the volume of the stomach.

Atrophic gastritis is described under Achylia gastrica.

**Morbid Anatomy of Chronic Catarrhal Gastritis.**—The mucous membrane is yellowish-gray or slate-gray in color, and in secondary catarrhal conditions produced by congestion may, in some parts, be intensely red. It is swollen, thickened, and covered with a closely adherent tenacious mucus, which is usually cloudy and gray in color, due to various cells, epithelia, and leukocytes, and occasionally it is tinged with blood. Enlarged veins, patches of ecchymosis, and small hemorrhagic erosions may be present. The mucosa in some instances forms papillary projections (etât mamelonné).

The pyloric portion is usually involved, though the inflammation may extend over the entire mucosa. The submucosa and muscular coats are at times hypertrophied or atrophied.

Microscopically the following conditions appear: The glands are enlarged, sacculated, and dilated in cyst-like forms, the tubuli in many places being atypic and branching like the fingers of a glove; the gland-cells are cloudy, granular, and in a condition of fatty degeneration, and the principal and parietal cells cannot be differentiated. Abundant small cell infiltration presses the glands apart, being especially marked toward the surface of the mucosa. Extensions of connective tissue may be seen passing from the mucosa between the glands. The mouths of the glands are at times filled with mucus which projects against the lumen. Mucoid transformation of the cells of the tubuli is a striking feature and may extend to the fundus of the glands and cells. Mucoid degeneration may replace the principal and parietal cells.

The mucus fills part of these cells, or may crowd the protoplasm and nucleus against the base, or rupture the cell-membrane and escape into the duct of the gland. The pyloric region is usually thus affected. Ewald has described this condition.

After a long period the inflammatory process may lead to a total destruction of the glandular layer, or atrophy of the mucous membrane of the stomach—Anadenia ventriculi (Ewald). Of this there are two forms, as heretofore noted:

(*a*) Phthisis ventriculi, atrophy of the stomach, or Anadenia ventriculi (Ewald), is a thinning of the coat of the stomach, with a retention of or usually an increase in the size of the organ (dilatation). There are fatty degeneration and destruction of the glands, the process progressing from the surface of the stomach inwardly. In the early stage no glands are found, but glandular cysts are present near the submucosa. Later these disappear and the mucous membrane consists chiefly of round cells. The submucosa is changed and the muscular layer is thinner.

(*b*) In cirrhosis, or sclerosis ventriculi, the stomach coats are thickened and there is a great reduction in the volume of the organ; in some cases it is no larger than a pear and the walls may be 2 to 3 cm. in thickness, the greatest increase being in the submucosa, *where the process starts*. The inflammatory process causes the formation of fibrous tissue, which progresses from the submucosa to the surface, spreading around the glands, constricting them, and finally replaces them with fibrous tissue. The hypertrophy extends to the muscular layer.

This condition may coexist in the cecum and colon and so be difficult to distinguish from diffuse carcinoma. Proliferative peritonitis with perihepatitis and ascites are at times associated.

Atrophy of the gastric mucosa is described under Achylia gastrica.

**Symptoms of Chronic Gastritis.**—These develop slowly; the appetite diminishes or is irregular or easily satiated. There is a feeling of fulness or pressure in the gastric region after eating. Occasionally *heartburn* or cardialgia in the epigastrium or precordial region or behind the sternum occurs, generally *at the height of digestion;* while with hyperchlorhydria it is present *on an empty stomach.* Discomfort or pain on pressure over the stomach is present. *Belching of gas is the most frequent symptom* and it is usually odorless; water-brash (pyrosis), a bitter or a tasteless fluid, may be brought up into the mouth (regurgitation); the stomach and intestines are often markedly distended with gas. Nausea is frequently present and occasionally vomiting.

When the latter occurs, it is frequent in the morning, when the stomach is empty, and consists chiefly of slimy mucus, and sometimes of partly digested food of the previous day with mucus. It may take place after breakfast. There is a sour, bad, or salty taste in the mouth. The patient in some cases complains of palpitation and shortness of breath (dyspeptic asthma or, more correctly, dyspnea). The pulse is small and sometimes slow. There are fulness in the head, insomnia, lack of energy, and distaste for work. Dizziness may be present. Sensation of fear, depression, melancholia, or hypochondriasis occur in some patients. The tongue is usually covered with a thick gray moist fur, though not always so, and it cannot be said to be characteristic. The margins are at times indented. Odor of the breath is present when there is disease of the mouth or teeth or atony of the stomach with fermentation. Headache is quite common and a desire to yawn. There is at times the so-called stomach-cough, doubtless due to pharyngeal irritation.

The patient's appearance may be quite good and he may preserve his weight. In severe cases he looks quite badly, and shows black rings under his eyes, has cold hands and feet, and chills easily. Some even lose considerable weight and become thin and emaciated. In the severe types with nervous symptoms, intestinal fermentation or putrefaction are often present and *auto-intoxication* is undoubtedly a factor. The bowels are, as a rule, *constipated;* though occasionally diarrhea or diarrhea alternating with constipation are present.

**Physical Examination.**—*Inspection.*—The gastric region appears bloated.

*Percussion.*—Tympanites is present, but the stomach is usually in the normal position.

*Palpation.*—The gastric region is sensitive to pressure; tenderness is rather diffuse. No real pain or sense of resistance.

*Splashing sound* can be produced if liquid and gas are present. It is only abnormal if found at a time *when the stomach should be empty.* It would then show atony. If found in an *abnormal position* (low), it is an evidence of dilatation or ptosis. With movable kidney ptosis can be diagnosed.

*Urine* is scanty; contains phosphatic and urate deposits. Specific gravity is increased.

**Diagnosis.**—The *presence of gastric mucus* in the stomach contents is the *chief diagnostic point* in chronic *gastritis*, so that examination of the *vomitus* or, *preferably, of the gastric contents* after a *test-breakfast is imperative.* The *diagnosis should not be made* from *clinical symptoms alone.*

*Gastric Contents.*—One hour after Ewald's test-breakfast or the one I employ—2 slices (60 grams) of bread without butter and 300 to 350 c.c. of water—the latter being slightly less than Ewald's, the contents of the stomach are withdrawn. The following are the conditions found present:

Total acidity is diminished; free hydrochloric acid is markedly lessened or is small in amount or absent; pepsin and rennet are present, but diminished; erythrodextrin present in small quantities;

achroödextrin and sugar abundant; quantity of gastric contents frequently normal (under 100 c.c.) or may be slightly more (100 to 150 c.c.), which last would show some motor insufficiency.

The pieces of roll are not as fine as normally, but larger and coarser. Mucus is usually intimately mixed with the food remnants and is adherent to the morsels of food. The stomach-contents are thick, tough, and sticky, and difficult to filter. A glass rod dipped into them and lifted up will draw up strings of mucus with it. Acetic acid added to the filtrate produces turbidity.

*Mucus that has been swallowed is never mixed with the food remnants*, but floats as isolated balls on the surface.

Mucus in some cases is in large amount, while there may be very little in others. In the latter event, lavage of the empty stomach will determine its presence, in shreds or flakes.

In the fasting condition there are often only a few cubic centimeters of turbid liquid in the stomach, consisting chiefly of mucus of an alkaline, neutral, or slightly acid reaction. If no contents can be thus secured, lavage again will show the mucus.

The vomitus shows the same characteristics already described, but the examination by the test-breakfast is more accurate.

*Microscopically.*—Mucus, round cells, and epithelial cells are found to be present. In doubtful cases the microscope may differentiate the types of mucus. If squamous epithelia be mixed with it, it probably comes from the mouth or pharynx; if pigmented alveolar epithelia, probably from the air-passages. Columnar epithelia mixed with mucus shows it is gastric.

With *acid gastritis* we find the total acidity and free hydrochloric acid slightly increased and the presence of mucus. I had recently a case in which the total acidity averaged 90+ and free hydrochloric acid 70+, with a large amount of mucus.

The so-called cases of mucous gastritis merely contain an excessive amount of mucus, with little or no hydrochloric acid.

With atrophic gastritis there is absence of hydrochloric acid, absence of pepsin, and absence of rennet, as described under Achylia gastrica.

If dilatation be present, we have the physical signs and gastric findings of such.

Einhorn finds small shreds of the mucosa present in the wash-water of some cases of chronic gastritis, due, as he believes, to erosions.

*Motor function* may be *normal* or slightly diminished, so that the ingesta escape before fermentation can occur This is the usual course in the ordinary type of case.

In some cases with hypertrophy the motor function may be increased. If atony or dilatation is present, there are motor insufficiency and fermentation.

In those with excessive mucus production, the action of the saliva and gastric juice is interfered with, and, though the motor function is good, the ingesta passes into the intestines with little change, and intestinal fermentation or putrefaction results.

*Absorption.*—This depends on the severity of the case; in milder cases, with the iodid of potassium test, it seems normal; in severe cases it is interfered with.

**Course.**—The duration of chronic gastritis is long, often extending over many years. Marked improvement may take place, but relapses are apt to occur from indiscretions. Milder cases can be permanently cured.

**Differential Diagnosis.**—*Chronic Gastritis.*—No severe pain; no circumscribed spot painful to pressure; no hematemesis; no cachexia; no marked emaciation, except in severe cases of long duration; free HCl diminished or absent; gastric mucus present; slow course.

*Ulcer of the Stomach.*—Hyperchlorhydria present, but not invariably so; severe pain in the epigastrium, with intervals free from pain when the stomach is empty; local tenderness which is circumscribed; dorsal pain, hematemesis, or occult blood in the stool or gastric contents; microscopic pus; *no mucus;* patient has appearance of suffering; no true cachexia.

*Cancer.*—Age, usually over forty-five; *rapid course;* free HCl usually markedly diminished or absent; lactic acid present; mucus sometimes present; pain generally continuous, but not as acute as in ulcer; Boas-Oppler bacillus; cachexia; tumor on physical examination; small amount of blood or occult blood present in gastric contents; microscopic pus; blood or occult blood in the stool; hematemesis much less than ulcer; foul odor to vomitus at times present.

*Achylia Gastrica.*—Slow course; scarcely any gastric juice; acidity very low (2+ to 4+); absence of HCl; absence of pepsin; absence of rennet; usually *no mucus;* no lactic acid. In the early stage (transitional) Riegel holds that mucus may at times be present.

**Achlorhydria Hæmorrhagica Gastrica.**—This is frequently a reflex from a diseased appendix or gall-bladder. Large numbers of bacteria, most frequently streptococci or colon bacilli, are present in the gastric contents. Free hydrochloric acid is absent. Occult blood from erosions is present in the gastric contents. The amount recovered after the test-breakfast is usually less than that ingested, and the bread is in a coarse state of division, practically as when swallowed, and thin mucus is incorporated with it. The contents when aspirated vary from a *yellow tinge to a light orange.* Total acidity is 6+ to 8+. Pain occurs in about one-third the cases directly after food, but may be present at other times. Vomiting of a sour taste occurs in about half the cases and nausea is frequent. These patients generally have periods of depression alternating with nervous excitement. Constipation is usual. Pylorospasm usually is present which, together with the erosions, often accounts for the pain. The condition is primarily in most cases a reflex disturbance of gastric secretion (absence of hydrochloric acid) with a secondary invasion of the stomach by microörganisms and ultimately erosions of the mucosa. It has also been found secondary to infectious diseases and other conditions, and cure of the primary disease has usually resulted in the cure of the gastric lesion. Pilcher[1] reports many such cases from Mayo's clinic.

*Gastric Neuroses.*—Symptoms not uniform; character of food makes little difference; indigestible food may be well borne and digestible food may cause symptoms; *no gastric mucus;* HCl may be diminished or in some cases normal or increased, and the gastric findings often vary at different times in the same patient. *Subjective symptoms are sometimes similar to chronic gastritis,* but they disappear and reappear abruptly.

**Prognosis.**—Some cases can be cured; many improved. Relapses may occur. The affection is frequently a tedious one.

**Treatment.**—This may be divided into: (1) Prophylaxis. (2) Hygiene. (3) Local treatment of the stomach. (4) Diet. (5) Mineral springs. (6) Medication.

*Acid Gastritis.*—The borderline cases, *acid gastritis,* should receive the treatment of hyperchlorhydria; also occasional lavage to remove mucus, say, twice a week, is advisable. In a recent case I have employed it daily, using several ounces of milk of magnesia (Phillips) to the quart of water.

Extract of belladonna, $\frac{1}{3}$ grain (0.022), t. i. d. before meals, and magnesia usta and soda bicarbonate, *āā,* 10 grains (0.6), combined with resorcin resub., 5 grains (0.3), in water, an hour after meals, as suggested by Theodorus Bailey, is excellent treatment. Milk of magnesia, 1 to 2 drams (4.0–8.0), is also of value.

Van Valzah and Hayes report cases.

1. *Prophylaxis.*—Unquestionably, repeated attacks of mild so-called acute dyspepsia (acute gastritis) may ultimately lead to the development of chronic gastritis. The causes of both conditions are practically the same. The patient, therefore, should masticate thoroughly, avoid bolting the food, overindulgence in alcohol, tobacco, very hot or very cold food and drink, indigestible food, etc. He should rest for fifteen to thirty minutes after meals before returning to business. The mouth and teeth should be kept in good condition; and if there are aural or nasal discharges escaping into the mouth, or tonsillar or pharyngeal inflammation, treatment should be instituted.

Cardiac disease should be treated with cardiac stimulants, especially if there is failing compensation; and diseases of the liver and kidney should receive appropriate diet and treatment, so as to lessen the chances of secondary gastritis.

2. *Hygiene.*—Slow eating, with subsequent rest; exercise, preferably in the open air, driving, golf, rowing, walking, and horseback—all of which should be carried out in a leisurely manner and not overdone, so as to exhaust the patient, are useful. Moderate gym-

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[1] Jour. Amer. Med. Assoc., Nov. 19, 1910.

nastic exercises five or ten minutes daily, as with an exerciser, with open windows, are of value.

Avoid badly ventilated rooms and sleep with the windows open.

Cold salt-water sponging morning and night, followed by friction with a rough towel, is of service.

If the patient is excessively nervous, change of climate may be necessary.

3. *Local Treatment.*—The removal of the mucus is of importance. This can be done by two methods: By administering alkaline remedies that will dissolve the mucus and by lavage.

In mild cases lavage is not always necessary, and the use of alkalis is of service.

They should be administered about an hour before breakfast and, if necessary, also before luncheon and dinner. For example, in 6 to 8 ounces (200 to 250 c.c.) of hot water, soda bicarbonate, 30 grains to 1 dram (2.0–4.0), or lime-water, $\frac{1}{2}$ ounce (16.0), or milk of magnesia (Phillips), 1 to 2 drams (4.0 to 8.0).

Magnesia usta, 30 grains to 1 dram (2.0 to 4.0), is also of use, alone or combined with equal quantities of soda bicarbonate.

Penzoldt has demonstrated that mucus will adhere to bismuth. Rose takes advantage of this and gives a tablet consisting of 10 grains (0.6) magnesia usta and 10 grains (0.6) bismuth subnitrate, to be chewed with a full glass of water one hour before meals; or two tablets, if required.

The magnesia usta, milk of magnesia, or soda bicarbonate are especially useful.

*Lavage.*—When the mucous secretion is more marked, lavage should be substituted; or if a mild case does not improve by the above method. This should be employed on the empty stomach before breakfast, so as to aid subsequent digestion. An alkali should preferably be added to the fluid to dissolve the mucus. The stomach should be washed with the patient both sitting and lying down and turning on the sides, to remove all mucus possible, and washed *until the outflow is clear.*

The following are excellent: Milk of magnesia (Phillips), 1 to 2 ounces (30.0 to 60.0) to the quart (liter); or lime-water the same quantity; or soda bicarbonate, 1 to 2 drams (4.0 to 8.0); or magnesia usta, 1 to 2 drams (4.0 to 8.0)—all to the quart (liter).

Normal saline solution—1 dram (4.0) salt to water 1 pint (500 c.c.)—or boric acid, 1 dram (4.0) to the quart (liter), may be employed.

I use normal saline solution combined, preferably, with milk of magnesia or magnesia usta.

Fleiner mixes 2 parts sodium chlorid and 1 part soda bicarbonate and employs 1 dram (4.0) to 2 to 3 quarts (liters) of water.

*Frequency of Lavage.*—Once a day before breakfast is often sufficient; in some cases it may be necessary to repeat it before supper.

If there is dilatation with fermentation, resorcin (resub.), 5 to 15 grains (0.3 to 1.0), or the same quantity of salicylic acid or sodium salicylate; or gomenol, 15 drops to ½ dram (1.0 to 2.0); or potassium permanganate, 1½ to 5 grains (0.1 to 0.3); or listerin, glycothymolin, or borolyptol, 1 dram (4.0)—all to the quart (liter), can be employed.

In such event, I wash with the alkali in the morning to remove mucus, and with the antifermentative at night.

In some cases, lavage with nitrate of silver (1 : 5000 to 1 : 2000) is of value, used every two or three days, preceded by warm water lavage, to first remove mucus. No saline should be used in the silver solution. Saline solution may subsequently be used if the silver cause pain or irritation.

Pepper, in place of this, advocates an aqueous solution of silver nitrate; dose, ⅛ to ¼ grain (0.008–0.016) internally, three times a day for a short period. This should be kept in a dark bottle.

*Electricity.*—Unless atony with lessened motor function of the stomach, or severe subjective symptoms, with *little mucus* are present, electricity is of no practical value.

When there is little mucus, it may aid to stimulate gastric secretion. In such event the intragastric method is preferable to the percutaneous. Removal of the mucus is of the first importance. Electricity, however, is of service applied to the abdomen to *increase intestinal peristalsis.*

*Massage.*—*Vibratory Massage.*—The same holds true of these methods. They are also of value over the intestines to promote peristalsis.

*Hydrotherapy.*—In sensitive cases hot water compresses applied to the stomach are useful.

4. *Diet.*—This is an *important feature*, and its character depends on the severity of the symptoms. It is always preferable to give four or five light meals than three meals full a day. In severe cases give food in liquid and semiliquid form (mushes), such as milk or matzoon with Vichy, kumiss, bacillac, lactone-buttermilk, barley, oatmeal, or rice soups prepared with milk; or chicken soup or bouillon, with raw egg beaten up; sanatogen (plain or flavored), somatose, plasmon, or tropon can be added to the soups.

Later, soft-boiled eggs, mashed potatoes, rice gruel, scraped raw or broiled beef, toast baked in the oven, stale bread (white), butter, cocoa, and weak tea.

The diet should be mixed, the albumin somewhat reduced; the carbohydrates should be given in suitable form, avoiding those that contain too much cellulose. Fats, such as butter and cream, are especially necessary, when there is *loss of nutrition.* In the latter case I feed by the " scales," endeavoring to put on weight, improve assimilation, and at the same time ameliorate symptoms.

The mere estimate of requisite calories and feeding by this method *alone* is of no value, as each patient is a rule to himself.

For example: In severe cases, give at first liquids and mushes.

8.00 A. M. Milk, one glass, with 2 ounces (about 125 c.c.) of lime-water, or peptonized milk, 8 ounces (about 250 c.c.), with sanatogen, 1 dram (4.0).

10.30 A. M. Matzoon and Vichy equal parts, in all 6 to 8 ounces (200–250 c.c.).

1.30 P. M. Oatmeal soup or chicken soup, with an egg beaten in, 8 ounces (250 c.c.).

4.00 P. M. Same as at 10.30 A. M.

6.30 P. M. Same as at 8.00 A. M.

9.30 P. M. Milk and Vichy equal parts, in all 8 ounces (250 c.c.).

Additions can gradually be made to this diet. Milk, however, does not agree with some, and soups and broths must be substituted.

The following diet is useful in many cases for a week or two, but must be modified to suit the individual:

| | | Calories. |
|---|---|---|
| 8.00 A. M. | 1 cup cocoa or tea, about two-thirds milk, approximately | 100 |
| | 1 lump of sugar | 40 |
| | 2 soft-boiled eggs | 165 |
| | 2 ounces zwieback, or toast, or stale white bread (2 slices) | 150 |
| | ½ ounce butter | 115 |
| 10.30 A. M. | 8 ounces (250 c.c.) koumiss, matzoon, or milk | 168 |
| | 2 ounces crackers or somatose biscuit | 150 |
| | ½ ounce butter | 115 |
| 1.00 P. M. | 2 ounces of steak, chicken, or chop | 70 |
| | 3 ounces of mashed potatoes or rice | 130 |
| | 2 ounces white bread (stale), or toast, or zwieback | 150 |
| | 1 cup tea, about two-thirds milk, approximately | 100 |
| | ½ ounce butter | 115 |
| 4.00 A. M. | 7 ounces (250 c.c.) milk, mixed with 1 ounce top-cream | 210 |
| | 1 ounce crackers | 100 |
| | ½ ounce butter | 100 |
| 6.30 P. M. | 8 ounces (250 c.c.) hominy, rice, or cereal boiled in milk | 450 |
| | 2 scrambled or poached eggs | 165 |
| | 2 slices bread (average about 2 ounces) | 150 |
| | ½ ounce butter | 115 |
| | | 2858 |

The above is for about three weeks, and the diet of each patient should later correspond, as nearly as possible, to the usual mode of living.

There are things which it is necessary to forbid, such as fried food; meat with tough fibers or that is too old or too fresh, pork, sausages; lobster, salmon; chicken salad; mayonnaise; cucumbers, pickles, corn, radishes, celery, cabbage; hot breads, brown and Graham bread; also fresh bread and *all* alcoholic beverages, which last I believe do special harm to the inflamed mucous membrane.

Foods which diasagree should be interdicted. Sugar should be taken in small quantity and avoided by some. Soda-water and candy are forbidden; also hot and cold drinks and ice-cream. Veal, as a rule, in this country is interdicted, as it is often tough.

Salt is of value, as it aids the production of HCl.

Beef, mutton, lamb, chicken, potatoes, hominy, rice, oatmeal, spinach, lettuce, asparagus, eggs, etc., are all admissible. Water should be taken in small amounts during the meal.

*Smoking.*—Excessive smoking should be stopped. It is chiefly the *tobacco juice* from chewing the cigar, carried by the saliva, that damages the mucous membrane of the stomach; chewing tobacco should be forbidden.

*If a cigar-holder or cigarette-holder be employed*, I can see no objection to two cigars or four cigarettes a day; otherwise it should be interdicted. If there is *nasopharyngeal catarrh*, smoking should be *stopped.*

5. *Mineral Waters.*—These dissolve the gastric mucus, hasten the emptying of the stomach, and often stimulate the mucosa. On account of the rest and regular life and diet the springs are often preferable, though the waters may be taken at home. The most useful are the saline and the saline-alkaline waters.

(*a*) *Saline Springs.*—These contain chiefly sodium chlorid and varying quantities of carbonic acid gas, and stimulate the secretion of hydrochloric acid. The most notable are Kissengen, Homburg, Wiesbaden, Soden, and Saratoga (Congress Spring). Dose, glass of mineral water on arising.

(*b*) *Alkaline-saline Springs.*—These contain sulphate of soda, sodium bicarbonate, sodium chlorid, and carbonic acid gas.

The Carlsbad Springs are the most famous. Marienbad and Saratoga (Hawthorne Spring) belong to this group, also Glauber's Salt Springs and Glauber's salts.

One can employ the imported Carlsbad salts or Galuber's salts and by adding sodium bicarbonate to Glauber's salts imitate imported waters more closely.

Wolff's formula for artificial Carlsbad salts:

| | | |
|---|---|---|
| ℞. | Sulphate of soda | 30.0 |
| | Sulphate of potassium | 5.0 |
| | Sodium chlorid | 30.0 |
| | Carbonate of soda | 25.0 |
| | Biborate of soda | 10.0.—M. |

Sig.—½ to 1 dram (2.0–4.0) in warm water before breakfast.

These springs are of value for the solution of large quantities of mucus and for constipation. Care should be taken not to purge the patient excessively, and nervous cases do not take them especially well.

6. *Medication.*—The methods described will often be sufficient, but medication is of service as an accessory.

Leube was the first to recommend the use of dilute hydrochloric acid to supplement this deficiency of the gastric juice. Ewald advised the use of large amounts, 40 to 60 drops, taken in divided doses three times a day after meals.

For example: The entire dose in a glass of water and commencing one-half hour after meals, a third of this being taken every fifteen minutes.

I prefer a smaller dose, from 15 to 20 drops, in a small glass of water three times a day one-half hour after meals, and taken in three divided doses, as advised by Ewald. This is impossible to pursue with some patients, as they will not devote the time; so in such an event a single dose can be administered, commencing at 5 drops and gradually increasing to 15 drops.

The following is an excellent prescription:

℞. Tinct. nucis vomicæ.......................... 12.0 (ʒiij);
Acid. hydrochlor. dilut. }
Comp. tinct. cinchona } ..................āā 16.0 (℥ss);
Aq. destil............................q. s. ad. 125.0 (℥iv).—M.
Sig.—1 to 2 drams (4.0–8.0) t. i. d. in water one-half hour after food.

A convenient method of administering hydrochloric acid is in the form of oxytin. This can be given in 5-grain (0.3) capsules. Ten grains of oxytin represents 5 minims of dilute hydrochloric acid. There is also a preparation of oxytin with nux vomica. A 5-grain (0.3) capsule of this preparation contains 5 minims of the tincture of nux vomica. One to two of these capsules can be given half an hour to an hour after meals.

*Pepsin is present in considerable quantity* in chronic gastritis, so it is hardly indicated; though some add $7\frac{1}{2}$ to 15 grains (0.5–1) in combination. Papayotin, papain or papoid, or pancreatin, 15 to $22\frac{1}{2}$ grains (1.0–1.5), with sodium bicarbonate, have been suggested; also the diastase combinations, wines of pepsin, etc.

The use of artificial digestants would tend to weaken the gastric functions, and are not indicated.

The bitter medicaments as stomachics, to stimulate the secretory function and appetite, are often employed, and Riegel believes the hydrochloric acid acts in this way. They should be used fifteen minutes before meals in 1 to 2 ounces of water.

Among such remedies are tincture aurantii amara; tincture amara (bitter tincture, Squibb's); tincture calumba; fluidextract calumba; tincture cardamomi; tincture hydrastis; tincture gentian compositum; fluidextract hydrastis; fluidextract condurango; fluidextract quassia.

The average dose for this purpose of any of these remedies would be from 15 to 20 drops; quassia, in 5 to 10 drops; and tincture nux vomica, if given as a stomachic, in 5 to 10 drops.

They may be given alone or in combination, with smaller individual doses.

The alkaloidal form of administering stomachics is often of value. Thus:

Condurangin (Abbott's), $\frac{1}{67}$ grain (.001).
Quassin (Abbott's), $\frac{1}{67}$ to $\frac{1}{12}$ grain (0.01–.005).
Condurangin (Merck's), $\frac{1}{10}$ to $\frac{1}{4}$ grain (.0065–.016).
Quassin (Merck's), $\frac{1}{32}$ to $\frac{1}{3}$ grain (.002–.02).
Hydrastin (Merck's), $\frac{1}{4}$ to $\frac{1}{2}$ grain (.016–.032).

Strychnin arsenici, $\frac{1}{100}$ grain (0.00065), and quassin, $\frac{1}{10}$ grain (0.0065), are a good combination.

Orexin, ½ to 1 grain (0.032–0.065), can be given in bouillon for the same purpose.

In the gastritis of phthisis, carbonate of creosote or guaiacol carbonate, 5 grains (0.3) each t. i. d. after meals, are of value.

*For Nausea and Vomiting.*—Cerium oxalate, 1 grain (0.065), alone or combined with soda bicarbonate or bismuth subnitrate, 2 grains (0.13), or any of the methods described under Acute Gastritis. Lavage may be necessary.

*For Belching.*—Milk of magnesia, ½ to 1 dram (2.0–4.0), or magnesia usta, 10 grains (0.6).

*For Gastric and Intestinal Fermentation.*—Resorcin resublimed, 1½ drams (6.0); aqua menthæ piperitæ, q. s. 4 ounces (125.0). Dose, 2 drams (8.0) t. i. d. after food in water, or ichthoform or ichthalbin, 5 grains (0.32) each, or sodium benzoate, 10 grains (0.6) t. i. d., etc.

The following prescriptions are also excellent, and are recommended by William H. Thomson:

℞. Resorcinolis.................................. ʒiij (12.0);
Tr. nucis vomicæ.............................. ʒiv (16.0);
Syrup zingiberis.............................. ℥ij (125.0);
Aq. menth. piperit.....................q. s. ad. Oss (250.0).—M.
Sig.—Two teaspoons in water, half an hour after meals.

℞. Potassii bichromatis.......................... gr. iss (0.096).
Bismuthi subcarb.............................. ʒiss (6.0);
Ext. gentianæ........................q. s. ad.—M.
Div. in pillulæ, xxx.
Sig.—One-half an hour before meals.

*For nervous symptoms associated with intestinal putrefaction* I would refer to the chapter on the latter subject. Iron and arsenic can also be added to the treatment.

*For Constipation.*—Patients under treatment with the Carlsbad waters require no treatment for constipation; otherwise attention must be paid to this condition.

The patient should have a movement every day, go to the closet at a definite hour, and endeavor to secure bowel action. This can be assisted by a 2-ounce injection of olive oil, or by a glycerin or gluten suppository, or 2 drams to ½ ounce (8.0–16.0) glycerin in 1 ounce (30.0) of water, by means of a small rectal syringe.

Green vegetables, such as spinach, asparagus, lettuce, green peas, etc., are of service.

Stewed fruits, such as prunes, apples, or pears, are often effectual.

Some cases do well with food containing much cellulose and with rye bread, but many cannot take them. A glass of cold or hot water on rising is of value.

If mild cathartics are necessary, cascara (fluidextract), ½ to 1 dram (2.0–4.0), or the aromatic fluidextract; or extract cascara, 1 to 2 grains (0.065–0.13); or regulin, 1 to 2 drams (4.0–8.0) at meals; or compound rhubarb pills—all at night.

The following prescriptions are of service:

| | | |
|---|---|---|
| ℞. | Aloin | gr. $\frac{1}{4}$ (0.016) |
| | Podophyllin | gr. $\frac{1}{6}$ (0.011) |
| | Atropin. sulph. | āā gr. $\frac{1}{100}$ (0.00065) |
| | Strychnin | |
| | Cascara ext. | gr. $\frac{1}{4}$ (0.016).—M. |

In one pill.

| | | |
|---|---|---|
| ℞. | Ext. aloes | āā gr. $\frac{1}{8}$ (0.008).—M. |
| | Ext. nucis vomicæ | |
| | Ext. belladonnæ | |

In one pill.

| | | |
|---|---|---|
| ℞. | Podophyllin | gr. $\frac{1}{6}$ (0.011) |
| | Ext. nucis vomicæ | āā gr. $\frac{1}{4}$ (0.016).—M. |
| | Ext. physostig. | |

In one pill.

| | | |
|---|---|---|
| ℞. | Aloin | gr. $\frac{1}{4}$ (0.016) |
| | Strychnin | gr. $\frac{1}{6}$ (0.00108) |
| | Ext. belladonnæ | gr. $\frac{1}{8}$ (0.008).—M. |

In one pill.

Other remedies are described under Constipation.

Olive-oil injections to be retained at night; an occasional enema of soapsuds, not over 1 quart (liter); massage or vibratory massage or electricity to the intestines are useful.

# CHAPTER XI

# ACHYLIA GASTRICA—ACHLORHYDRIA HÆMORRHAGICA GASTRICA

## ACHYLIA GASTRICA

(*Synonyms*.—Atrophy of the Stomach; Atrophy of the Gastric Mucosa; Anadenia Ventriculi; Phthisis Ventriculi; Atrophic Gastritis; Catarrhus Atrophicans.)

**Definition.**—Achylia gastrica (the term first suggested by Einhorn) may be defined as a functional perversion of the stomach, characterized by the absence of the gastric secretion (of hydrochloric acid, pepsin, and rennet).

**Introduction.**—In many cases, atrophy of the mucosa is the cause, and the condition is permanent. Einhorn, however, has reported a case in which there was eventually some return of secretion, so that portions of the mucosa could not have been much altered; and a case of achylia in a vegetarian, apparently an atrophy from disuse. Achylia gastrica may result from organic changes in the stomach, or may be a pure neurosis, so I prefer to place it in a special chapter.

The *loss of function may be temporary from nervous disturbances*, and A. Rose[1] and the author[2] have observed achylia occurring in gastroptosis, with a return of secretion following the cure of ptosis. Temporary achylia is present occasionally with mucous colic.

**History.**—Atrophy of the gastric mucosa was first described in connection with pernicious anemia by S. Fenwick,[3] and later by Lewy,[4] Ewald,[5] Osler,[6] Kinnicutt,[7] Nothnagel, Boas, and others. It was believed to be the cause of pernicious anemia and productive of the fatal result. Herter has demonstrated the influence of intestinal putrefaction, chiefly through the gas bacillus (Bacillus aërogenes capsulatus), in the production of pernicious anemia, and favorable results produced by intestinal irrigation and lactic acid fermented milk diet. Stockton has called to our attention that achylia does not occur in the early stages of pernicious anemia, but only when it becomes severe. In view of these facts, achylia seems to be a secondary and not a primary cause.

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[1] Atonia Gastrica, Rose and Kemp.

[2] Observations on Dilatation of the Stomach and Gastroptosis, Medical News, August 6, 1904. Mucous Colic, American Medicine, March 4, 1905.

[3] Lancet, July, 1877.

[4] Berlin. klin. Wochenschr., 1887, No. 4.

[5] Ibid., 1886, No. 32.

[6] Amer. Jour. Med. Sci., vol. xci, 1886, p. 498.

[7] Ibid., 1887, p. 419.

Numerous non-fatal cases of achylia have been reported by Ewald,[1] Boas, Einhorn,[2] Jaworski, Jones, and Martius, and the latter has written a work on the subject.

**Etiology.**—Severe chronic catarrhal gastritis, or toxic gastritis, may produce permanent destruction of the glands; atrophy from disuse, as in vegetarians; achylia, associated with cirrhosis of the liver (syphilis); or with carcinoma of the stomach, or, rarely, with carcinoma of other organs; occasionally with diabetes mellitus; achylia with pernicious anemia. Organic changes are present in these cases. Achylia may occur *as a temporary functional disturbance* in nervous conditions, neurasthenia, gastroptosis, and mucous colic. Achylia may also be secondary to a general infection, such as typhoid fever,[3] etc.

It is quite common in the middle and later years of life, but has occurred in a number of young persons, especially in the transitory type.

**Morbid Anatomy.**—When the achylia is of nervous origin, or associated with gastroptosis or mucous colic, there are no organic changes in the mucosa, and the condition is a temporary functional disturbance of secretion. Rose and myself believe the achylia in mucous colic due to the gastroptosis and not to the nervous condition.

Einhorn has shown that in cases where a portion of the mucous membrane has been aspirated, showing the organic changes of achylia, there has been some return of secretory function under treatment, so that this is no evidence of permanent destruction of secretory power in all the gastric glands.

Of the organic changes, there are the two types described under the terminal stage of chronic gastritis:

1. Phthisis ventriculi—round-celled infiltration and fatty degeneration, with thinning of the stomach wall; round cells taking the place of the glands; and the stomach is normal in size or may be dilated (Fig. 154).
2. Cirrhosis, or sclerosis ventriculi, a fibrous inflammation, starting in the submucosa, fibrous tissue takes the place of the glands, and the stomach is contracted and the walls thickened.

**Symptoms.**—One can scarcely say that there are symptoms characteristic of achylia gastrica; it is *the examination of the gastric contents* which will alone determine the true diagnosis.

In describing the symptoms it seems best to classify achylia under certain groups, some of which we may dismiss briefly:

1. Gastroptosis (splanchnoptosis), with its symptoms; gastric disturbances; achylia is present in some cases.
2. Mucous colic, with its symptoms; occasional presence of achylia.

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[1] Berliner klin. Wochenschr., 1892, Nos. 20 and 27.
[2] New York Med. Presse, Sept., 1888.
[3] Stockton, Amer. Jour. Med. Sci., Aug., 1909.

3. Patients with no symptoms and enjoying good health. Einhorn calls attention to this class, one of which had the habit of rumination, and achylia was found present, of forty years' duration. Patient had no other symptom. Clinically this class cannot be considered.

4. Cases with gastric symptoms of varying severity, associated with intestinal disturbances. *These are the most common type.*

There are loss of appetite, a feeling of fulness or pressure in the epigastric or gastric regions, and in some cases severe paroxysms of pain, usually soon after eating and persisting for some time; vomiting may occur soon after the ingestion of food; *belching of gas;* headache and occasionally vertigo; usually constipation.

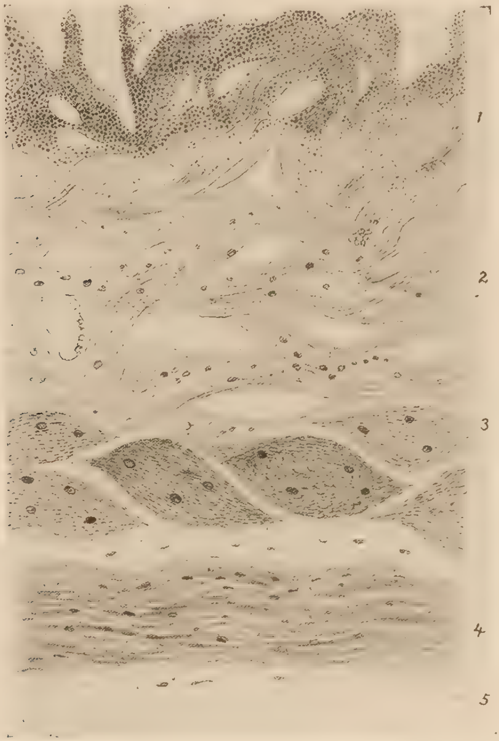

Fig. 154.—Achylia gastrica: 1, Mucosa; 2, submucosa; 3 and 4, muscularis; 5, serosa; section shows round-celled infiltration of mucosa and absence of glands.

Some cases may remain fairly well nourished. In others there may be considerable loss of weight, which extends over a period of several years, and nervous symptoms may be present.

In cases in which nutrition is preserved, the intestines perform the digestive functions of the gastro-intestinal tract.

Einhorn describes cases whose symptoms resemble hyperchlorhydria, with pains one to two hours after eating, which are relieved by food and drink, especially the latter, which prevents irritation of the mucosa by the coarse particles. This has been given as an explanation for the pain.

5. Cases with marked intestinal disturbances, especially diarrhea, or occasionally diarrhea alternating with constipation; quite frequently there may be no gastric symptoms or slight belching or a feeling of pressure.

Some of the cases lose considerable weight and strength and feel weak. I have recently treated a case suffering from these symptoms who was, in addition, markedly nervous. At the end of four months' treatment gastric secretion returned and all symptoms disappeared, there being 15 pounds increase in weight—a case of nervous achylia.

Some cases of this type may present symptoms (subjective) of diabetes, according to Einhorn.

6. Cases with severe anemia (described by Riegel) with diarrhea. Examination of the blood shows secondary anemia, and of the stomach, achylia gastrica. Diarrhea favors the production of anemia and the latter improves under treatment. These cases must not be confounded with pernicious anemia. The blood examination easily differentiates.

7. Achylia developing during acute febrile conditions, such as influenza (Ewald). Riegel believes it probably preëxisted, and that the intestines, which formerly performed the digestive functions, are thus disturbed and achylia symptoms first appear in consequence.

8. Patients suffering from nervous conditions, or neurasthenia, or gastric neuroses; achylia a temporary perversion.

My case under the diarrheal class belonged to this type.

9. Pernicious anemia with achylia, in which the blood-findings are typic; megalocytes and nucleated red blood-corpuscles (normoblasts and megaloblasts), etc.

**Diagnosis.**—The diagnostic feature of achylia is the gastric analysis one hour after Ewald's test-breakfast. The characteristics are as follows:

Total acidity is 2+ to 4+, or even neutral; HCl = O; pepsin = O; rennet = O (rennet zymogen may be present); propeptone = O; peptone = O; lactic acid = O; or faint trace; erythroödextrin = O; sugar = +; gastric contents have no odor, no evidence of fermentation; quantity of liquid is very small, aside from that soaked into the particles of bread (this is quite characteristic); bread particles are not minutely minced, but rather coarse; absence of mucus.

Schmidt and Riegel believe mucus to be present in some cases in the early stage of achylia, developed from chronic gastritis, while there are some glands remaining in mucoid degeneration, and that the presence of mucus does not signify that the case is not one of achylia. Some cases of old chronic gastritis certainly seem on the borderline.

The small amount of fluid in achylia is explained by the fact that the liquid chyme rapidly leaves the stomach, leaving the solid particles, and that practically no secretion takes place in the stomach.

*Motor function* is often normal or even increased. It is diminshed where there is degeneration with dilatation of the stomach.

*Absorption.*—Though this has apparently seemed normal by the iodid of potash test, in some cases it is evident that this test is often fallacious, in view of the fact of the general loss of nutrition in some patients.

I agree with Riegel that from the pathologic condition present, absorption must be diminished.

**Course and Prognosis.**—In nervous achylia, secretion may be resumed in a few months under proper treatment, and in gastroptosis and mucous colic the condition is dependent on the treatment of these diseases.

In some patients the condition perhaps exists for years, with the patient in good health; while in others the subjective symptoms may be removed or cured, while the objective symptom (the analysis) persists. Others run a long and protracted course.

With pernicious anemia and carcinoma the prognosis of achylia depends on the primary disease.

**Diagnosis.**—Repeated analyses of the gastric contents are necessary to arrive at the diagnosis.

| | *Achylia Gastrica.* | *Cancer of the Stomach.* |
|---|---|---|
| Gastric contents: | Little fluid, no mucus, low acid reaction (2+ to 4+), no HCl, no lactic acid, coarse particles, no blood. | Mucus, acidity higher, lactic acid, free HCl may be present, though usually absent, contents more fluid, and odor and food less coarse, blood or occult blood present. |
| *Stool:* | No blood. | Blood, or occult blood. |
| *Tongue:* | Often clean. | Coated. |
| *Pain:* | At times. | Constant. |
| *Motor power:* | Normal usually. | Diminished. |
| *Course:* | Slow. | Rapid. |
| *Loss of weight:* | Gradual. | Rapid. |
| *Cachexia:* | None, or slow emaciation. | Rapid and peculiar type. |
| *Tumor:* | None. | Present later. |
| | With achlorhydria hæmorrhagica gastrica there are numerous bacteria and occult blood in the gastric contents, as shortly described. | With chronic gastritis there is much mucus in the gastric contents and pepsin is present. |

The possibility of achylia gastrica being the cause of *various types* of gastro-intestinal disturbances, of chiefly intestinal derangement or irregularities, or of severe anemia must be considered. Its association with pernicious anemia, gastric neuroses, various nervous symptoms, gastroptosis, and mucous colic must be remembered. These facts further emphasize the importance of gastric analysis.

**Treatment.**—This depends on the cause. Rose's belt, if achylia is due to gastroptosis or mucous colic, is necessary. If associated with nervous affections, these conditions should receive treatment. Such patients should have their nutrition improved, and placed on the diet shortly to be laid down. As in these cases achylia is a functional disturbance, the secretion should be stimulated by—

Strychnin, $\frac{1}{60}$ to $\frac{1}{30}$ grain (0.00108–0.0021), t. i. d. before meals, or condurango (fluidextract), 15 to 20 minims (0.88–1.18), or tincture nux vomica, 5 to 10 minims (0.29–0.59), with the addition of hydrochloric acid, thus:

| | | |
|---|---|---|
| R. | Tinct. nucis vomicæ<br>Acid. hydrochlor. dilut. | āā ʒiij (12.0) |
| | Comp. tinct. cinchona | ℥j (30.0) |
| | Pure pepsin | ʒiss (6.0) |
| | Aq. destil. | q. s. ad. ℥iv (125.0).—M. |

Sig.—Two drams in water t. i. d. before meals (preferably).

Pancreatin, 5 to 10 grains (0.3–0.6), with soda bicarbonate, 15 grains (1.0), holadin or pancreon, 5 grains (0.5), given three times a day, are useful to aid intestinal digestion, or taka-diastase, 5 grains (0.33), can be given after meals in cases with diarrhea to aid digestion of starchy food. Cellasin tablets, 5 grains (0.3), t. i. d., are also useful.

In the general treatment of achylia gastrica endeavor: (1) To stimulate gastric secretion. (2) To arrange the diet so that the food is easily digested.

To *stimulate gastric secretion* employ stomachics, such as nux vomica, condurango, etc., fifteen minutes before meals, as just described. The occasional use of lavage twice a week is of service in some cases. Litten advises the use of a 2 per cent. dilute hydrochloric acid solution for this purpose.

Von Noorden has employed hepptin (the natural gastric juice of the pig) with success in some cases, in tablespoon doses, three times a day. It can be given after meals in water, seltzer, or tea. If administered in milk, it will coagulate the same if it is warm, but it can be taken by this method. Oxyntin (a hydrochloric acid preparation in powder form) can be given in capsules, 5 grains (0.3), each alone or with nux vomica. Dose, one to two capsules three times daily before meals. They can be secured already prepared.

Intragastric faradization (preferably) twice a week or percutaneous faradization may be valuable. I would not advise this in the purely nervous cases.

If the symptoms simulate hyperchlorhydria, the use of water, 8 ounces (250 c.c.), or crackers and milk one to two hours after meals, as suggested by Einhorn, lessens the irritation.

*Diet.*—This is very important. Food should be prepared so as to pass readily from the stomach, and should be finely divided and thoroughly masticated. Meat should be given in small quantity, finely chopped, scraped (rare or broiled), chicken, pigeon, raw scraped beef, calves' brain, steak, fish, and game.

Starchy foods are excellent and should be thoroughly prepared; membranes covering any such should be removed. Starch is rapidly converted into sugar.

Mushes and liquids are preferable. Pea and bean soup (strained), barley, oatmeal, rice, sago, and potato soups. Mushes from rice, potato, tapioca, oats, flour, etc.; puddings. Soft-boiled eggs; yolks of eggs in soups; raw eggs beaten with water or milk; koumiss; matzoon; bacillac; bread; crackers; milk, plenty of cream and butter are indicated.

Somatose, sanatogen (plain or flavored), tropon, and meat powder; Valentine's or Liebig's beef-juice; Armour's extract of beef can be added.

Cocoa possesses considerable nutritive power. Tea and coffee well diluted with milk can be given. My personal view is *that alcoholic beverages are objectionable,* though some allow them in moderation.

The second indication in the feeding is to employ it at frequent intervals, about every three hours, in small quantities, but so that the sum total shall be considerable. The object should be rather to overfeed and to increase the patient's weight by selecting those foods which best agree in each case.

In diarrheal cases, constipating food, such as potatoes and rice, should be selected. Feed by the " scales " to increase body weight is my rule in cases where loss of weight has occurred.

For the preparation of meat powder, either of the following methods is excellent:

(1) *Debove's Method.*—Roast finely chopped lean beef on a tin-plate until desiccated, then powder in a mortar.

(2) *Einhorn's Method.*—Dry finely chopped meat three to six hours on a stove; then pound in a mortar and grind twice in a coffee-mill.

Butter, milk, cream, eggs (soft boiled or raw), potatoes, rice, peas, and beans (strained through a colander), cocoa, chocolate, and a small amount of beef, chicken, or game, and crackers or stale bread always seem to agree. I have often given 4 to 8 raw eggs a day For example:

| | | Calories. |
|---|---|---|
| 8 A. M. | Oatmeal with cream, 150 gm. | 400 |
| | Cocoa with milk, 200 gm. | 135 |
| | Soft-boiled eggs (2) | 160 |
| | Toast (2 slices) | 160 |
| | Butter, 20 gm. | 163 |
| 11 A. M. | 6 ounces (200 c.c.) milk with raw egg | 240 |
| | Crackers, 2 oz. (60 gm.) | 150 |
| | Butter, 20 gm. | 163 |
| 2 P. M. | Soup, bean or pea, 100 gm. (with 1 egg and 10 gm. of cane-sugar) | 122 |
| | Rare meat scraped, 100 gm. | 215 |
| | Mashed potatoes, 50 gm. | 63 |
| | Spinach, 50 gm. | 30 |
| | Bread, 2 slices (60 gm.) | 135 |
| | Butter, 20 gm. | 163 |
| | Tea and milk (2 lumps of sugar) | 60 |
| 5.30 to 6 P. M. | Soft-boiled egg (1) | 80 |
| | Rice, 50 gm.; milk, 200 gm. | 302 |
| | Bread (1 slice) | 67 |
| | Butter, 20 gm. | 81 |
| | Tea and milk | 60 |
| 9 to 9.30 P. M. | 6 ounces (200 c.c.) milk or koumiss | 128 |
| | Zwieback, 50 gm. | 129 |
| | Butter, 20 gm. | 163 |
| | Total calories | 3269 |

It may be necessary to modify this diet and give less, especially at first.

In cases of severe anemia, rest in bed; arsenic in large doses, beginning with Fowler's solution, 5 minims (0.296), t. i. d. and increasing to 10 to 15 minims (0.59–0.88) t. i. d.; or by hypodermic in sterile water in the form of atoxyl, $\frac{1}{3}$ to $\frac{2}{3}$ grain (0.022–0.044), every other day, and, in addition, peptomangan (Gude), iron tropon, or any other good iron preparation.

Intestinal irrigation is of value in these cases when *putrefactive processes* are present in the intestines, for example, with 1 to 2 ounces (30.0–60.0) peroxid of hydrogen or acetozone 15 grains (1.0) to the quart (liter) of water. Enteroclysis is especially advocated in pernicious anemia cases. Matzoon, koumiss, buttermilk, bacillac, lactone buttermilk are suitable as a diet, and lactic acid bacilli tablets internally for the intestinal putrefaction present in this disease.

I have found urotropin, 5 to 10 grains (0.3–0.6), combined with equal quantities of benzoate of soda, is of great service as a remedy for intestinal putrefaction.

Resorcin and the other remedies suggested for this purpose under Chronic Gastritis and in the paragraph on Indicanuria are of value.

The bismuth preparations, notably bismuth subcarbonate or subnitrate, 10 to 30 grains (0.6–2.0) t. i. d., can be given if diarrhea is present, and in this type boiled milk, potatoes (mashed), and rice are serviceable.

If there is atonic dilatation of the stomach, lavage and the treatment for this condition must be carried out and Rose's belt applied.

## ACHLORHYDRIA HÆMORRHAGICA GASTRICA

**Definition.**—Achlorhydria hæmorrhagica gastrica is characterized by the absence of hydrochloric acid in the gastric secretion, usually the result of reflex disturbance from disease of some other organ, or from some other primary disease, and by the presence of gastric erosions, with occult blood, mucus, and a large number of organisms, most frequently streptococci, colon bacilli, or diplococci, in the gastric contents.

**Introduction.**—This condition has been extensively investigated by the Mayos of Rochester in a large series of cases. In seven instances a reflex achylia gastrica (absence of all the secretions) was present. The disease is somewhat difficult to classify, though it appears usually to be the result of a reflex disturbance of gastric secretion, with a secondary infection of the mucosa. From the presence of mucus in the gastric contents it might be allied to a chronic gastritis of an infected type following a reflex disturbance of gastric secretion.

J. T. Pilcher[1] reports 271 cases from a large mass of material, or approximately one in every fifteen stomachs examined after the Ewald test-meal. As a further demonstration that achlorhydria hæmorrhagica gastrica is a reflex disturbance, it proved interesting to note the disappearance of all gastric symptoms after removal of the primary irritant, such as the appendix, gall-stones, etc.

**Etiology.**—In 156 out of 271 cases of achlorhydria hæmorrhagica gastrica reported, the onset of the gastric symptoms seemed to bear an immediate relation to other diseases; to infectious diseases in 38 cases; to circulatory disturbances in 12; to postoperative development in 14; and to derangement of the ductless glands in 24 cases. In 100 cases an operation was performed while achlorhydria hæmorrhagica gastrica was present. The appendix was involved in 36 cases; the gall-bladder, in 32; the gall-bladder and pancreas, in 16, and in the stomach there were 16 cases in which there was some additional lesion. There were 12 cases in which the appendix and gall-bladder were together diseased. Reflex nervous phenomena are doubtless primarily responsible for the inhibition of the production of hydrochloric acid in these cases, for it is a well-known fact that irritation in distant organs may produce an extraordinary degree of gastric disturbance.

**Morbid Anatomy.**—In 64 cases the stomach and pylorus were apparently normal. In 24, pylorospasm was demonstrated on the operating table. It accompanied appendicitis in 18 cases and gall-bladder involvement in 6. In 4 cases previous gastro-enterostomies were cut off, as they seemed not to be required. In several cases other lesions of the stomach were also present. In 2 cases, ulcer; in 1, pyloric insufficiency; in 3 the pylorus was much thickened;

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[1] Pathological Laboratories of St. Mary's Hospital, Rochester, Minn., and Jour. Amer. Med. Assoc., Nov. 19, 1910.

there was 1 congenital stenosis of the pylorus and 1 hour-glass contraction.

**Microscopic Examination.**—In 3 cases specimens of the gastric mucosa were secured for examination. There were non-contiguous erosions or ulcerations extending down to the muscular coat (Fig. 155) and a submucous infiltration of round cells (Fig. 156). In many places there was a marked engorgement of the capillaries due to the inflammatory reaction. There were masses of yellowish blood pigment lying irregularly over the epithelium which are not found in the normal organ. It was believed that they were probably due to precipitation of the ferruginous element contained in the blood-serum which had exuded from the eroded and inflamed areas.

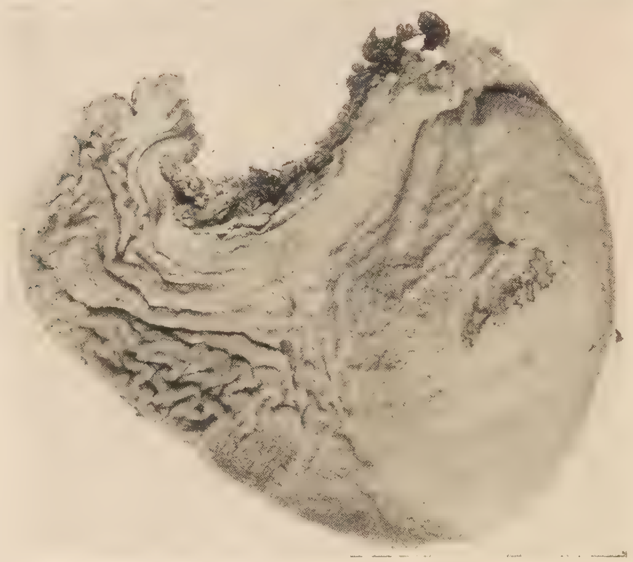

Fig. 155.—Gross appearance of posterior wall of stomach, showing non-contiguous erosions or superficial ulcerations in fundus (J. T. Pilcher).

**Bacteriology.**—These cases are characterized by the presence of a large number of pathogenic organisms in the stomach, particularly streptococci and colon bacilli. Their presence in great numbers is believed to be due to the lowered acidity of the gastric juice (absence of hydrochloric acid). The examination of 350 specimens from 150 stomachs showed the following results: Streptococci in 127 cases, colon bacilli alone in 8, streptococci and colon bacilli in 64, diplococci (marked) in 84, lactic acid bacilli in 42.

Staphylococci, the proteus, and leptothrix were always present. In several cases the cells of the mucosa possessed phagocytic properties. Degenerated leukocytes were almost always present, sufficient to be designated as pus in 48 cases. The bacteria were actively growing. Streptococci are apparently the most important factors, since they occurred in larger numbers in those cases in which pus was present. Probably the active irritation accounts for the presence of the erosions, and the latter are responsible for the pain. The *yellow color* in the gastric contents and the occult blood reaction must be due to the *oozing of serum and blood* from the *erosions*.

**Gastric Analysis.**—The results of analysis of the gastric contents are *quite characteristic*. The Ewald test-breakfast with water (not tea) is administered, and an hour later the contents are withdrawn. The amount recovered is usually less than that ingested. The bread is in a coarse state of division, practically as it has been swallowed. There is no viscosity, but the bread is frequently incorporated in thin mucus. The *color* of the contents is *quite characteristic*—varying from a *yellow tinge*, just off the white, to a *light orange*. Occasionally there is a slight rancid odor. The filtrate is clear and may have a yellow tinge. Remnants of former meals are seldom found, and if present are due to a continuous pylorospasm. The biuret reaction is usually present and also milk coagulation, showing there is no true "achylia." In 7 cases, however, "achylia" was present.

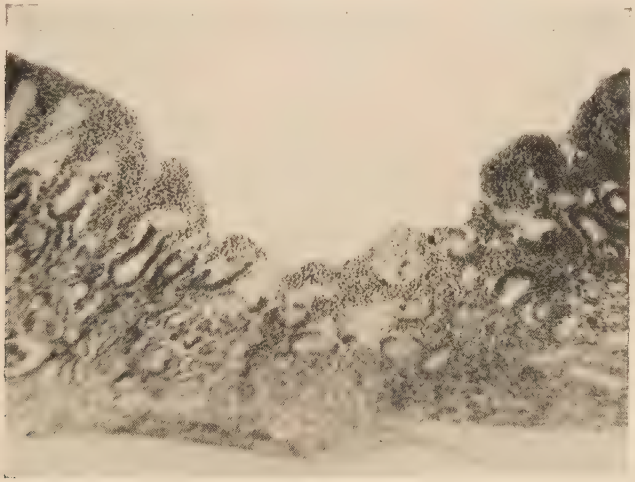

Fig. 156.—Photomicrograph through defect in mucosa in Fig. 155, showing destruction of epithelium and round-cell infiltration of submucosa (J. T. Pilcher).

*Free hydrochloric acid is absent* and combined acids are practically negative, though traces have been found.

Lactic acid is present in about 15 per cent. of cases. The Strauss method of determining lactic acid the investigators deem preferable, as by that of Uffelmann the reaction is usually obtained.

**Blood.**—The *reaction for occult blood is always present*. Pilcher employs the modified guaiac test, but the benzidin test is satisfactory also.

**Symptoms.**—The symptoms are somewhat inconstant and capricious, but these features are suggestive of the diagnosis when considered with the other facts. In the majority of cases there is oppression or distress in the epigastrium, amounting usually to a sensation of pain. This is generally of a burning, gnawing character, and occurs in about one-third of the cases immediately after the ingestion of food, and acids increase the pain. Sometimes the pain comes on much later or even before meals. It is usually increased by taking food, though it may be temporarily relieved thereby. Vomiting, particularly, relieves the pain, as do also alkalis and lavage. There is considerable belching, and sour, bitter, acrid eructations occur, probably due to the formation of organic acids. In about half the cases vomiting of the ingesta occurs immediately after meals, and the vomitus has a sour, bitter taste. Nausea is quite frequent, and hematemesis has occurred in a few cases. Constipation is the rule, but it may alternate with periods of diarrhea of from one to four weeks' duration. In some cases the bowel movements may occur directly after eating. The appetite is usually impaired, though vagaries are noted, and these patients discriminate as to the quantity and quality of their food, usually first discarding meats and fats. There is generally a loss of weight of from 15 to 25 pounds. These patients generally present a perverted mental attitude, having periods of depression alternating with nervous excitement. This mental condition is probably due more to worry over their disturbed digestion or to the fear of cancer than to a neurosis. The patient suffers from malaise, is easily fatigued, is unable to work, has occasional headaches, and at times insomnia.

**Diagnosis.**—The diagnosis of achlorhydria hæmorrhagica gastrica is usually not particularly difficult, as it generally *results as a reflex from primary disease in some other organ.* The determination of gallbladder disease, appendicitis, pancreatic disease, or some other primary disease is at once suggestive, as are the restoration of hydrochloric acid and the disappearance of occult blood after operation for a surgical condition extraneous to the stomach, or after the cure of some other medical condition. The absence of hydrochloric acid, presence of occult blood mucus, and the large number of organisms, streptococci, colon bacilli, etc., in the gastric contents, and the other peculiarities of these contents as already described are pathognomonic.

*Gastric Ulcer.*—Free hydrochloric acid, in the acute cases in excess, is usually present. Examination of the gastric contents shows *no mucus* and the *large number* of various types of bacteria. Bacteria, however, are present. The gastric contents do not present the peculiar appearance of achlorhydria, and in the latter the total acidity is very low, 6+ to 8+, and free HCl is absent. At times, however, one meets with cases of gastric ulcer with an absence of hydrochloric acid, and the possibility that an erosion of achlorhydria hæmorrhagica gastrica may become an ulcer must be considered. Under Gastric Ulcer, the writer refers to the view that gastric ulcer does not result from an erosion. In the case of achlorhydria hæmorrhagica, however, there are erosions plus an infection with various organisms, notably colon bacilli. The experiments of Türck show that by feeding dogs with pure cultures of colon bacilli one may produce gastric ulcer. Hence, it is possible that an erosion of achlorhydria hæmorrhagica gastrica may develop into an ulcer. In fact, Mayo refers to 2 cases of gastric ulcer associated with this condition. The type of gastric ulcer with absence of hydrochloric acid would in some cases seem to be the probable result, such as Connors' case, reported in chapter XII, page 255.

**Carcinoma of the Stomach.**—The age of the patient, rapidly progressive emaciation, and anemia associated with the gastric symptoms are suggestive. There is more *usually absence of free hydrochloric acid* (though in some cases in the early stage it may be present to even considerable amount). Lactic acid and Boas-Oppler bacilli are present. They may be absent in the *early stage* if HCl is present. The gastric contents do not present the peculiar appearance of achlorhydria hæmorrhagica gastrica, though blood or occult blood is present. There are not the numerous varieties of bacteria in excess. The glycyl-tryptophan reaction is sometimes an aid to diagnosis of carcinoma. There is usually disturbance of the motor functions of the stomach in cancer. With achlorhydria hæmorrhagica there is some other primary cause. Carcinoma may, however, develop from achlorhydria hæmorrhagica gastrica from an erosion.

*Chronic Erosions.*—There are the characteristic pieces of mucosa found in the wash-water. Usually chronic gastritis is present, though occasionally hyperchlorhydria. The peculiar yellow appearance of the gastric contents is absent, and there are no excessive numbers of various bacteria. The condition is not a reflex from some other primary disease.

*Chronic Gastritis.*—This condition may be differentiated from achlorhydria hæmorrhagica gastrica, though the latter might be considered an infective gastric catarrh resulting from reflex achlorhydria with infection. With chronic gastritis there is not the peculiar yellow appearance of the contents from serum exudation, the acidity is usually somewhat higher, and combined hydrochloric acid is generally present. There are not the excessive numbers of bacteria, and occult blood is usually absent and not *always present* as it is in achlorhydria hæmorrhagica. Chronic gastritis is not a reflex from another primary disease.

**Prognosis.**—In the cases reported, 172 have been traced as to the ultimate results. Sixty-one have entirely recovered, 38 have an occasional complaint, 23 feel comparatively well, 9 were moderately improved, in 26 there was no improvement, 4 became worse, and 11 died. Among those who died, the chief primary factors were pancreatitis, nephritis, myocarditis, and pernicious anemia.

One case developed carcinoma of the stomach, and two histories suggested the same condition, but were not positively confirmed.

The best *results secured* were in those operated on for the *primary cause*. One must remember, therefore, the possibility of gastric ulcer or carcinoma developing from an erosion of achlorhydria hæmorrhagica gastrica. The somewhat rare condition, phlegmonous gastritis, the author believes may occasionally result from this infected type of achlorhydria. The cure of the latter may, therefore, be prophylactic against more serious conditions.

**Treatment.**—The primary factor upon which the reflex achlorhydria depends should be determined and receive appropriate treatment. The most favorable results have been secured when this has proved to be a surgical condition amenable to cure, such as appendicitis, gall-stones, etc. Complete restoration of the hydrochloric acid, disappearance of the occult blood from the gastric contents, and cure of the gastric symptoms have resulted. If the primary cause be a medical condition, it should also receive treatment.

The secondary condition, achlorhydria hæmorrhagica gastrica, should be properly cared for.

*Diet.*—The diet is important, milk with lime-water or peptonized, koumiss, bacillac, fermillac, lactone buttermilk, gruels, broths, and raw eggs beaten up with milk are indicated during the first few weeks. Flavored sanatogen, somatose, plasmon, or tropon can be added to the gruels or milk. It is preferable at first to give four or five small feedings daily. Later, soft-boiled eggs, mashed potatoes, thickened gruels, rice well boiled and passed through a colander, scraped raw beef, stale toasted bread, etc., may be added. Fats, such as plenty of butter and cream, are necessary to increase the weight. Later there may be a gradual return to full diet of a simple type. Care should be exercised for some time. Acids cause pain from the erosions. Pickles, spices, alcohol, and all irritating material should be avoided. Smoking should be interdicted. Lobster, cucumbers, corn, sausage, hot breads, and all indigestible food should be avoided until complete recovery.

*Medication.*—Alkalis combined with bismuth give the best result. They dissolve or remove the mucus and the bismuth coats the erosions. One can combine soda bicarbonate, 15 grains to ½ dram (1.0–2.0), with equal doses of bismuth subnitrate, given stirred up in a half glass of water t. i. d. before meals. The addition of olive oil given ½ to 1 ounce before the other two meals helps nutrition, coats the erosions, and aids bowel action. Tonics are indicated, such as iron, tropon, or any good preparation. In view of the frequent presence of colon bacilli in the stomach, particularly when the achlorhydria is secondary to appendicitis or gall-bladder disease, treatment for colon bacillus infection, the writer believes, is indicated. The urine should be *examined also for these bacilli.* The writer advocates the administration of urotropin, 30 grains to 1 dram (2.0–4.0) daily, in 10-grain doses, combined with sodium benzoate, equal quantities, for achlorhydria hæmorrhagica gastrica. Vomiting is relieved by oxalate of cerium, etc. Lavage affords most rapid relief.

*Lavage.*—Lavage affords great relief to the pain and clears off the mucus. Mild antiseptics should be added on account of the bacterial infection. Acetozone, 1 : 1000 in normal salt solution, once daily is useful, and if there is much pus it could be employed more frequently, say twice a day. In some cases substituting lavage twice a week with silver nitrate solution, 1 : 5000 to 1 : 2000, followed by washing with normal saline solution; on the other days use acetozone. Tincture of belladonna in 10-drop doses several times daily, in some cases pushed to physiologic symptoms, is of value to relieve the pain from the erosions and also for pylorospasm.

In addition, one can employ for the pain hot fomentations or dry heat over the epigastrium. Orthoform hydrochlorid or anesthesin 5 to 7½ grains (0.3–0.5) t. i. d. can be given for pain. Lavage also affords relief. Cocain *should never* be used. Opiates should be avoided unless all other means fail, and should only be given under the immediate direction of the physician, first, codein, ⅛ to ½ grain (0.008–0.032), or, as a last resort, morphin, ⅛ to ¼ grain (0.008–0.016), by hypodermic.

Constipation or diarrhea should receive appropriate treatment.

## CHAPTER XII

# HEMATEMESIS—ULCER OF THE STOMACH—EXULCERATIO SIMPLEX—ACUTE AND CHRONIC EROSIONS—PERIGASTRITIS AND PERIGASTRIC ADHESIONS

### GASTRIC HEMORRHAGE

VOMITING of blood cannot be considered to be a proof of gastric hemorrhage, as it may come from the esophagus, nose, or mouth, or be coughed up and swallowed.

On the other hand, gastric hemorrhage may occur, and the blood only appear in the stools.

The causes of gastric hemorrhage may be classified as follows:

1. Trauma over the stomach; injuries to the mucous membrane from foreign bodies, as bones or needles; damage from the stomach-tube, mineral acids, or caustic alkalis.

2. Thrombosis or embolism of the vessels; aneurism; varicosities; atheroma of a vessel, or fatty degeneration.

3. Venous stasis due to cirrhosis of the liver; tumors of the liver; pylephlebitis; compression of the vena cava.

In case of cirrhosis the hemorrhage is from the mucous membrane or from esophageal varices.

4. Lesions of the heart or lungs, causing stasis in the vena cava.

5. Constitutional diseases, as leukemia; pseudoleukemia; pernicious anemia; hemophilia; scurvy; purpura; melena (morbus maculosus neonatorum).

6. Menstrual type, when amenorrhea is present.

7. Lesions of the central nervous system (brain or spinal cord).

8. Hysteria.

9. Ulcer of the stomach and carcinoma.

10. Acute infectious diseases, as yellow fever, scarlet fever, measles, small-pox, pneumonia, etc.

11. Weil's disease (epidemic jaundice); malignant jaundice (acute yellow atrophy); diseases of the pancreas.

12. Banti's disease with enlarged spleen, anemia, ascites, cirrhosis of the liver, gastro-intestinal hemorrhage, etc.

13. Phosphorus-poisoning.

14. Acute jaundice with hemorrhage, following operation.

15. Erosions—postoperative hematemesis due to these; the French describe them after appendicitis as vomito-negro-appendiculaire.

16. Exulceratio simplex (Dieulafoy), or superficial ulceration of the stomach.

17. Achlorhydria hæmorrhagica gastrica; hematemesis from the erosions has occasionally been reported. It is, however, rare.

As it is the general tendency to impute cases of gastric hemorrhage chiefly to ulcer or cancer, it seemed desirable to classify all causes.

**Symptoms.**—The chief symptoms are hematemesis and melena. Acute anemia develops if much blood is lost; the patient feels dizzy and weak and faints easily. The sight is blurred, pulse rapid and feeble, and extremities cold; rarely convulsions and death follow. Nausea and vomiting occur. The blood may be dark in color or coffee ground in appearance, or light if in a large amount. An evanescent rise of temperature may occur after the hemorrhage.

At times the symptoms may take place with no hematemesis and nothing definite visible in the stool, when Weber's or the benzidin test may be necessary to determine the presence of occult blood.

**Prognosis.**—The prognosis is rarely fatal from the hemorrhage itself, but depends on the primary disease.

**Treatment.**—A hypodermic of morphin, $\frac{1}{4}$ grain (0.016), and locally the ice-bag; extract ergot, $\frac{1}{2}$ dram (2.0) in solution by hypodermic, or ernutin, 5 minims (0.3) by hypodermic, and in addition gelatin 10 per cent. solution, or Trémoliere's solution gelatin, 5 per cent. with calcium chlorid, 2 per cent. These gelatin preparations should be given from 2 drams to 1 ounce (8.0–30.0) every half hour to an hour by mouth.

Tannic acid, 5 grains (0.33), or lactate or chlorid calcium, 15 grains (1.0), should be given in solution (water 6 ounces or 200 c.c.) by rectum; lactate of strontium or magnesium, 15 to 30 grains (1.0–2.0) in 2 ounces (60 c.c.) of water, can also be administered by hypodermic.

Adrenalin chlorid (1:1000); 5 to 10 drops (0.291–0.582 c.c.), by mouth or hypodermic is recommended, but it at times too rapidly increases pulse tension, especially if given hypodermically.

Hypodermoclysis or the rectal injection of normal saline at 120° F. are useful. Proctoclysis is of value. Ice-water lavage in rare instances may be necessary. Stimulants, such as strychnin, $\frac{1}{60}$ grain (0.00108), or camporated oil, $7\frac{1}{2}$ grains (0.5), camphor in 20 minims (1.3) of sterile almond oil, by hypodermic, may be required.

## ULCER OF THE STOMACH

(*Synonyms.*—Ulcus Ventriculi (Simplex); Peptic Ulcer; Ulcus Ventriculi Rotundum; Perforating Gastric Ulcer; Cruveilhier's Disease.)

Ulcer of the stomach is characterized by a destruction of the mucous membrane of the stomach varying in degree, exhibiting no tendency to heal, and in typic cases attended with gastric symptoms associated with pain, vomiting, and hematemesis. It was first described by Cruveilhier in 1829.

**Etiology.**—*Postmortem and Geographic Distribution.*—Brinton found evidences of gastric ulcer in 5 per cent. of persons dying from all causes, and most frequently in London and on the Continent. Others claim that gastric ulcer is found in at least 10 per cent. of cases. Gerhardt notes its frequent occurrence in Thuringia, and Von Sohlern its rarity in Russia, the Rhine region, and in the Bavarian Alps, believing this to be due to the vegetarian diet (rich in potassium salts) in these countries. This theory lacks scientific confirmation.

*Sex.*—Gastric ulcer occurs more frequently in women than in men. Welch places it at 60 per cent. in women and 40 per cent. in men, while Brinton believes it twice as frequent in women.

*Age.*—Cases have been reported in children under ten years. It occurs most frequently between twenty and forty years in females, and in males quite often between forty and fifty. Ewald places the highest mortality between forty and sixty. It may occur in old people.

*Occupation.*—Cooks, shoemakers, and procelain makers are most liable to this disease, but it seems a matter merely of coincidence.

*Etiology of Gastric Ulcer.*—Many theories have been advanced as to the cause of gastric ulcer, and C. F. Martin[1] describes, in an article on gastric and duodenal ulcers, some thirty-six causes. If one investigate the writings of various authors, this list can be considerably increased.

*Traumatism.*—Simple trauma probably cannot produce gastric ulcer unless other conditions are associated. Blows, falls, and the swallowing of foreign bodies, such as knives by jugglers, etc., have produced severe damage to the mucosa of the stomach and marked hemorrhage, without the ultimate production of ulcer. Griffini and Vassale have resected or burned out portions of the mucosa of the stomachs of animals and perfect healing has taken place, with no ulceration. Traumatism may be a factor, as is shown in the following case of mine: Girl, age twenty-two, with no gastric symptoms, was thrown from a trolley car, striking on the epigastric region. Pain and tenderness were present at this point and gastric symptoms developed. There was no visible hemorrhage, but pain and symptoms continued for six weeks, apparently of hyperchlorhydria, but local tenderness persisted at the point of injury. The patient then had a sudden gastric hemorrhage of severe type and the ultimate course was of ulcer of the stomach. Cure resulted in eight months, the case being observed for several years subsequently, with no recurrence. This girl was anemic before the accident and probably hyperchlorhydria was present, though no symptoms were complained of. A hematoma was, I believe, produced in the stomach wall, interfering with its nutrition, and the other conditions favored ulcer development. The patient also had a hematoma of the thigh from the same accident. Traumatism under such conditions may be a factor.

Anemia and chlorosis may predispose to ulcer, and Riegel and

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[1] Osler's Modern Medicine, v., p. 175.

Charles Stockton have shown that hyperchlorhydria frequently accompanies these conditions, and that it has an influence in the prevention of the cure of ulcer or even in its production. Experiments on animals have been performed for the purpose of studying the etiology of ulcer. Quincke and Daettwyler made animals anemic by venesection and produced lesions in the gastric mucosa. Section of the spinal cord, with the introduction of $\frac{1}{2}$ of 1 per cent. hydrochloric acid solution in the stomach of a dog, has produced ulceration (Koch and Ewald). It has occurred after injury to the anterior corpora quadrigemina.

Halliburton[1] states that just as poisons from without stimulate the cells to produce antitoxins, so harmful substances produced within the body are provided with *antisubstances* capable of neutralizing their effects. For this reason the blood does not clot within the blood-vessels, and Weinland recently has shown that there is formed in the gastric mucosa an antibody, an antipepsin, which opposes the digestive action of the acid gastric juice. If the antibody is deficient in a certain area, this unprotected region is readily injured by the gastric juice. The same writer holds that the intestinal epithelium forms an antitrypsin. The principle just referred to has been lately put into practical use. E. C. Hort[2] maintains that gastric ulcer is not a disease *sui generis*, but merely a local expression of a general dyscrasia caused by the presence in the blood of cytolitics for the gastric epithelium; he attempts by his treatment to reëstablish a condition of immunity of the gastric mucosa to the action of gastrolytic toxins and enzymes, which first cause and then render permanent the ulcers. He employs fresh normal horse-serum, given by mouth. The method and dosage are described later. Rosenau has produced gastric ulcer by the injection of diphtheria toxin. Botton[3] has produced gastric ulcer in animals by injecting the macerated gastric mucosa of other animals and of animals of other species.

The neurotrophic theory has been held by some. Stockton believes that nerve perturbation analogous to herpes may be a factor; and de la Verdora produced ulcer and hyperchlorhydria by injecting alcohol into the splanchnic and celiac plexus of a dog. Section of the vagi below the diaphragm has caused ulceration of the gastric mucosa. These experiments suggest that the nervous system may be a factor.

Kaufmann believes that lack of gastric mucus (amyxorrhœa gastrica) has a relation to hyperacidity and gastric ulcer.

Silberman introduced substances into the blood producing hemolysis (hemoglobinemia) with resulting anemia, and found that defects of the mucosa healed tardily. Türck has produced gastric ulcer by feeding dogs with pure cultures of the bacterium coli commune. R. T. Morris[1] holds that gastric ulcer may result secondarily from gall-bladder or from appendical infection with the colon bacillus, and Pilcher believes it may develop from an infected erosion from achlorhydria hæmorrhagica gastrica. It is known that burns of the abdomen may produce gastric ulcer, though usually the duodenum is affected.

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[1] Kirkes' Physiology, 20th Ed., p. 488.

[2] The Lancet (London), Dec. 21, 1907; Ibid., Feb. 15, 1908; Brit. Med. Jour., Oct. 10, 1909; Ibid., Jan. 5, 1910, p. 75.

[3] The Lancet, 1908, i, pp. 1330–1333.

Pavy held the theory of diminished alkalinity of the blood, but this can hardly be accepted. Erosions have been considered by some, notably Bassler, to be the cause of ulcers, but Langerhans opposed this from his experience in autopsies, and Einhorn claims that gastric erosions are a clinical entity and that ulcer does not result. The Mayos report 2 cases substantiating Pilcher's view. We know that autodigestion of the gastric mucosa occurs after death. The effect, therefore, of circulatory disturbances of the blood-vessels of the stomach in the development of ulceration is important.

Virchow first suggested that ulceration may result from the plugging of a nutrient artery to part of the mucosa by a thrombus or embolus, and that the infarction is destroyed by the gastric juice. Panum supported this view by producing infarcts and ulcers of the stomach in a dog by injecting an emulsion of wax into the femoral artery. Occlusion of the portal vein or of some of the large veins of the stomach may cause gastric ulcer.

Injection of chromate of lead into the gastric and splenic arteries (Cohnheim) has produced ulcer. Talma, by increasing the tension of the gastric wall by ligating the orifices of the stomach, has brought about ulceration. Artificial anemia by faradization of the stomach has caused ulcer. *Local interference with the circulation*, with *resulting necrosis*, associated with hyperchlorhydria and changes in the blood, are probably the most frequent causes.

Hyperchlorhydria is present in about 95 per cent. of the acute cases, but occasionally there is subacidity or achylia gastrica, as reported by Einhorn and others. Moreover, Spriggs[2] has demonstrated that the total acid content may be increased by hypersecretion, by interference with the motility of the stomach due to pyloric spasm, for example, and by the production of organic acids. This statement as to hyperchlorhydria in gastric ulcer refers, of course, *chiefly to the acute cases*, especially in the younger subjects. On the other hand, the reports from Mayo's cases show that of 75 per cent. of the cases that come to the operating-table, many of the patients do not show the high degree of acidity as is commonly supposed. Thus, in 250 cases of gastric or duodenal ulcer[3]

| Free HCl. | Below normal. | Normal. | Above normal. | Absent. |
|---|---|---|---|---|
| | 23 cases. | 102 cases. | 112 cases. | 13 cases. |

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[1] He believes toxins may also cause vascular disturbance in the terminal arteries with resulting ulcer.

[2] Brit. Med. Jour., May 21, 1910.

[3] New York Med. Jour., Sept. 4, 1909, Graham and Guthrie.

I have recently seen a case with very low acidity (Connors' case). Frequent gastric analysis showed free HCl trace or absent; lactic acid present; *microscopic pus* and occult blood. Multiple ulcers (non-malignant) were found by John Connors at operation. Probably gastric ulcer is not always produced by the same factors, and several of the theories described may apply. Hyperchlorhydria undoubtedly has a bearing on many cases, and frequently anemia or chlorosis.

**Morbid Anatomy.**—The peptic ulcer is found in the regions exposed to the gastric juice, in the stomach, lowest part of the esophagus, and upper duodenum. It is round or oval, occasionally oblong; is funnel shaped, the upper part being the larger; is of variable depth, its floor being formed by the submucosa, muscular tissue, serosa, or by adjacent adherent organs. The acute ulcer is usually small, punched out, with clean cut edges and a smooth floor, with no thickening of the peritoneal coat; occasionally the floor may be covered with a thick green or brown mucus (Fig. 157).

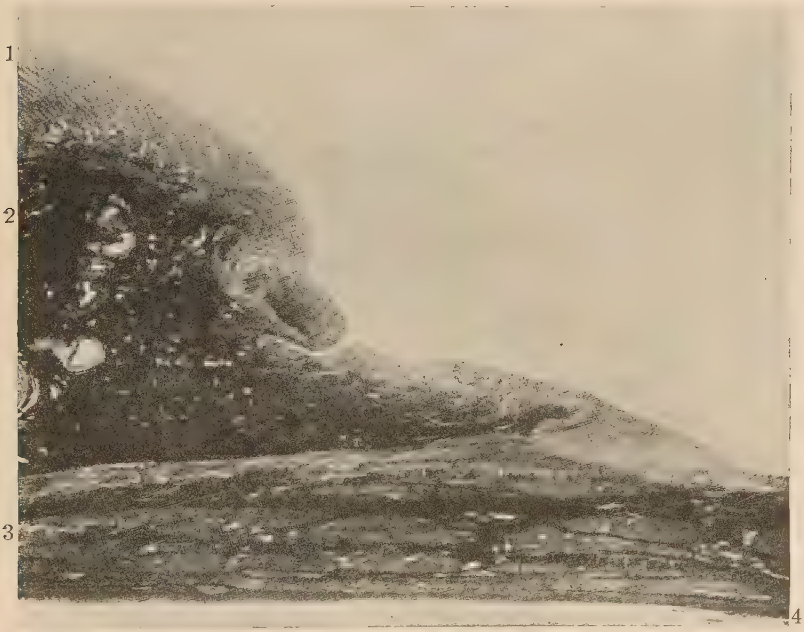

Fig. 157.—Gastric ulcer, acute: 1, Glandular layer; 2, submucosa; 3, muscular layer; 4, peritoneum. (From Bassler's "Diseases of the Stomach and Upper Alimentary Tract." Copyright, 1910, by F. A. Davis Company.)

The chronic ulcer is of larger size, with callous margins, and the border may be sinuous. It is often markedly indurated, so if situated at the pylorus it may feel like a tumor on palpation.

Embolism or endarteritis of the artery supplying the ulcerated region has been found, or a small aneurism on the floor of the ulcer.

*Microscopically.*—The ducts of the glands are cut off toward the base, being eaten away or digested up to where the tissue offers sufficient resistant power to the gastric juice.

Healing occurs by proliferation of the connective and glandular tissue near the margin of the gland. As the connective tissue contracts the proliferation of the glands is stopped. If the stomach wall is adherent to an adjacent organ and the ulcer perforates, the neighboring tissue may grow into the hole and unite with the connective tissue growing from within. Muscle-fibers do not regenerate. This is true of the large deep ulcers; the mucosa and muscularis roll in and adhere to the adjacent organ. There is further proliferation of tissue caused by irritation of the gastric juice, and the latter may cause erosion of vessels.

The rest of the *mucous membrane of the stomach,* as a rule, *remains normal.*

**Extent of the Ulcer.**—It may vary from the size of a pea to a diameter of 5 or 6 inches; the average being from a 5-cent piece to a 25-cent piece. Peabody reports one measuring 19 by 10 cm.

**Location.**—It is commonly situated on the posterior wall of the pyloric end of the stomach, at or near the lesser curvature. Welch states that out of 793 cases, 288 were in the lesser curvature, 95 at the pylorus, 96 on the anterior wall, 50 at the cardia, 29 at the fundus, and 27 on the greater curvature. Other statistics are given.

**Number.**—In about 80 per cent. of cases 1 ulcer is found; in a trifle over one-half the remainder, 2 ulcers; in the balance, 3 to 5 ulcers; Osler reports 34 ulcers in 1 case, and Lange 1 in which he could not count them.

**Progress of the Ulcer.**—1. Cicatrization may occur, with formation of a connective-tissue scar, which tends to depress and contract. Depending on its location, it may cause stricture of the pylorus, esophagus, or an hour-glass stomach. In other situations it may produce no trouble.

2. Progressive necrosis may take place and there may result:

(*a*) Erosion of a blood-vessel with severe or occasionally fatal hemorrhage from perforation of a large vessel, such as the gastric, hepatic, or splenic artery, portal vein, etc.

(*b*) Adhesions to neighboring organs or various perforations. The stomach may become adherent to the liver, gall-bladder, spleen, pancreas, or intestines, and there may be perforation into these organs.

If the ulcer is on the anterior surface, then direct perforation and general peritonitis may follow.

There is sometimes a circumscribed peritonitis when adhesions form with other organs, and a local abscess, which may later perforate into the peritoneal cavity.

The ulcer may perforate into the lesser peritoneal cavity and cause subphrenic abscess; the diaphragm may be perforated and a communication formed with the pleura, lungs, pericardium, and even with the left ventricle; or, rarely, an emphysema of the subperitoneal tissue occurs, which may pass into the posterior mediastinum; or adhesions may form and a perforation of the anterior abdominal wall take place; or general emphysema of the subcutaneous tissues may rarely result.

In cases with adhesions to adjacent organs, with or without abscess, localized growing tumors, hard in consistency, may be formed, and Gerhardt notes the possibility of mistaking these for carcinoma, but the history and gastric analysis make the diagnosis.

**Symptoms.**—Some cases present typic symptoms, in which event the diagnosis is easy; others suffer apparently from a simple hyperchlorhydria for a long period of time, with no special symptoms pointing to ulcer; while in others the condition is latent.

In the *latent cases* the patient is sometimes apparently perfectly well, when there will suddenly develop hematemesis or symptoms of perforative peritonitis. Others may not vomit, but suddenly turn faint and weak, become pale with a feeble pulse, presenting the *symptoms of internal hemorrhage.* Examination of the stool for occult (concealed) hemorrhage by Weber's, the aloin, or the benzidin test is a valuable aid.

*Cases Simulating Hyperchlorhydria.*—Kaufmann,[1] of New York, has suggested that in cases of hyperchlorhydria not yielding to treatment and in whom the pain is of a gnawing or tearing character the suspicion of ulcer is justified. This is undoubtedly true, and repeated examinations of the gastric contents and stool for blood or occult blood are indicated. Pus in the gastric contents is significant.

*Typical Case.*—As a rule, the symptoms of gastric ulcer develop slowly, and are as follows: at first a feeling of fulness and presssure after eating; gradually increasing to pain in the epigastrium, which may become so severe that the patient is afraid to eat. Nausea, regurgitation, or vomiting may occur early.

Pain occurs *generally a few minutes after eating*, though sometimes one-half to one hour later, and persisting during digestion. If there is hyperacidity, milk or the white of eggs (raw) may temporarily relieve the pain. Liquid nourishment causes the least disturbance. Coarse substances, hot ingesta, and large quantities of food increase it. It is of a burning or gnawing character. Epigastric pain is increased on pressure and the sensitive point is usually circumscribed.

A few weeks later dorsal pain begins, gnawing in character, lying to the left of the spine, between the eighth and tenth vertebræ, alternating with the epigastric pain; at times there is sensitiveness on pressure in this region.

The epigastric pain is not continuous, as in cancer, but there are periods of relief. Later, vomiting may occur, one to two hours after meals, of very acid, watery material, mixed with food; *emesis generally relieves the pain*; occasionally there is vomiting late at night or early in the morning if gastrosuccorrhea (hypersecretion) is associated with ulcer.

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[1] New York Med. Jour. and Philadelphia Med. Jour., March 11, 1905.

Appetite is variable; at times the patient desires food, but fears to eat on account of pain; constipation is generally marked; amenorrhea is frequent in women; anemia marked in many cases.

These symptoms continue, then hemorrhage suddenly occurs, and is visibly present in one-third to one-half the cases, and in a very large percentage *when occult hemorrhage* is included, as it should be. W. L. Rodman,[1] holds that hemorrhage is a marked symptom in at least 50 per cent. of all cases, and if the gastric contents and stools were carefully examined macroscopically, microscopically, and for occult blood from day to day, *the probabilities* are that blood would be *found in nearly every case*. With this last the author *cordially agrees*.

(1) *Hemorrhage may be occult*, no vomiting of blood, the patient turning pale and faint and in a cold sweat, and on the next day there are tarry stools, or occult blood is found in the stool by Weber's or the benzidin test. Progressive anemia may be caused by small repeated hemorrhages.

(2) The patient may experience a sense of fulness after a meal and become nauseated and restless; then hematemesis occurs in large amount, of fluid blood, bright red, or of liver color, brown, or coffee grounds, mixed with food.

Patient may feel faint, extremities cold, temperature subnormal, become collapsed, and, rarely, even die from hemorrhage. Convulsions and unconsciousness may precede death. Death from internal hemorrhage can occur without vomiting. Blood is generally passed by the stools (melena), black and tarry in color, and may be found in cases with no vomiting or in latent cases.

Convulsions from cerebral anemia, or hemiplegia from thrombosis, or amaurosis (possibly permanent) have occurred. A temporary rise of temperature may follow the collapse. Persistent temperature shows complications. The vomitus usually shows hyperchlorhydria and no mucus. With chronic ulcer hyperchlorhydria may not be present. Tongue is clean and red, rarely coated.

Often there are remissions and exacerbations of the symptoms, and they may be protracted; from no apparent cause a relapse may occur. With an unhealed ulcer, complications or perforation may occur at any time. In others, ulcer symptoms may diasappear, but those of gastric dilatation, adhesions, etc., may follow. In the long cases, from pain and self-starvation, marked emaciation takes place, the suffering shows in the patient's face, but there is not the sallow appearance of cancer. Tetanic attacks complicating chronic ulcer have been reported by Kaufmann.[2]

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[1] Jour. Amer. Med. Assoc., Sept. 15, 1906, vol. xlvii, pp. 842–845.

[2] Amer. Jour. Med. Sci., April, 1904.

*Pain.*—The epigastric pain usually occurs about the center of this region, in the median line, just below the tip of the ensiform. Occasionally it is more to the right or left, and lies in a circular area of 1 to 2 inches in diameter. Throbbing and pulsation may be felt in the epigastrium. Pain is *usually increased on pressure*, rarely lessened. It is injurious to frequently manipulate the *painful area* or subject it to *marked pressure*. Singer[1] calls particular attention to an early sign, which consists of *discomfort* or *pain* radiating from the epigastrium *toward* the costal arches, and thence along the intercostal nerve-roots to the spine. The regularity of the appearance of this pain or sense of discomfort, especially in connection with eating, is characteristic even when there is scarcely any dyspeptic disturbance. Testing with the algesimeter is not advisable.

In place of vomiting, some patients regurgitate acid chyme, with pyrosis, or suffer from nausea.

*Motor Function.*—In uncomplicated cases of gastric ulcer this is increased. If pyloric stenosis or adhesions complicate, then the function is interefered with.

*Examination of the Stomach Contents.*—If the diagnosis of ulcer has been positively determined, it is preferable not to pass the tube. If there is no recent hematemesis, or if vomiting is excessive, it is well to give the test-meal and examine, in addition, for occult blood. For example, in order to combine the test both for motor function and chemic analysis, the night before a meal of meat, potatoes, rice, a few dried raisins, and some chopped spinach can be given. On the following morning the Ewald test-breakfast can be administered and the contents withdrawn an hour later. The presence of raisins, rice, and spinach withdrawn at this time give valuable data. The Mayos[2] wash out the stomach and examine the washings for food remnants. Moreover, while the stomach-tube is still *in situ*, the organ is artificially distended with air by means of attaching a Davidson's syringe, and it is then carefully palpated for tumors or ridges. It is deflated before removal of the tube. If hemorrhage is recent, the writer does not advise this method of inflation. Lavage after the contents are removed is of service, as it checks emesis, and thus renders a hemorrhage less liable to recur.

If the patient states there has been a hemorrhage, but the diagnosis is in doubt, the tube should not be passed earlier than ten days to two weeks after the history of hematemesis. The stool can be examined at once for occult blood.

If vomiting occurs, the vomitus may be examined by preference, but the quantity and quality of the food and time of ingestion would influence the analytic findings and might lead to error. Immediate examination of the *vomitus and stool for occult blood is important.*

In about 95 per cent. of acute uncomplicated cases of ulcer, hyperchlorhydria is present; the total acidity is high, from 90+ to 150+, and free hydrochloric acid 50+ to 60+ or even 90+, and *there is no mucus.* The Mayos hold that of 75 per cent. of cases coming to operation many do not show high acidity. These are chiefly chronic cases.

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[1] Medizin klinik, Berlin, Dec. 18, 1910, vi, No. 51.

[2] New York Med. Jour., Sept. 4, 1909 (Graham and Guthrie).

There are cases of subacidity, or even of achylia gastrica, with ulcer.

*The Absence of Hyperchlorhydria Does Not Exclude the Presence of Ulcer.*—In all *doubtful cases* gastric analysis and examination *for occult blood and pus* in the *stomach-contents and stool* should be carried out.

In some cases we must think of the possibility of development of carcinoma on the base of a chronic ulcer, but in such event a recent exacerbation of symptoms which were previously less marked, loss of weight, weakness, and increasing anemia, occur. Cachexia and appreciation of tumor on palpation occur later.

*Urine.*—The quantity is reduced when there is much vomiting and food is diminished. There are no characteristic changes.

**Complications.**—Perforation and general peritonitis; circumscribed peritonitis; sacculated abscess; adhesions with other viscera, with or without perforation of them; pyloric stenosis; stenosis of the cardia; perigastritis with adhesions; subphrenic abscess; perforation of the diaphragm, pleura, lungs, pericardium, or heart; hour-glass contraction of the stomach; external fistulous opening; anemia of severe type; mediastinal emphysema, and subcutaneous emphysema may occur.

Stenosis of the pylorus, due to stricture or hypertrophy from spasm, produces dilatation of the stomach and its symptoms; stenosis of the cardia causes dysphagia and regurgitation of food.

*Perforation.*—This occurs in ulcers on the anterior stomach wall, or from perforation of a circumscribed abscess, with resulting general peritonitis. The symptoms are sudden pain, at times with a tearing sensation, shock, muscular rigidity, rapid distention of the abdomen; tenderness on pressure; disappearance of liver dulness; cold sweat; rapid and feeble pulse, followed by a rise of temperature, singultus; frequently vomiting; anxious and sunken face (facies Hippocratica); usually coma; then death. Leukocytosis, especially increase in the polynuclears, is present.

In perforation, with circumscribed abscess formation, the symptoms are less intense and are localized. Perforation of the stomach occurs usually after a full meal, or following coughing, sneezing, or local mechanic violence.

If adhesions form with other organs, these may be perforated.

*Frequency of Perforation.*—Brinton gives the frequency of perforation as 1 in 8 cases. Others place it at 6 to 7 per cent.

In women, Brinton places one-half the perforations at the age of fourteen to thirty, the average being twenty-seven; while in men they are distributed up to fifty, the average age being forty-two.

The chances of perforation of an ulcer on the anterior stomach wall are 5 to 1 in its favor, on account of its mobility, which prevents adhesion formation, but ulcers are much less frequent in this location.

Subphrenic abscess (pyopneumothorax subphrenicus, when gas is present) may occur.

*Etiology.*—The chief causes are as follows: Posterior perforating ulcer of the stomach (the most frequent cause); traumatism of the liver; abscess of the liver; retroverted appendix, and perforation of a duodenal ulcer.

The boundaries of the abscess-cavity are: above, the diaphragm; below, the stomach and liver; to the left, the spleen; to the right, the suspensory ligaments of the liver. The liver is pushed down and the diaphragm upward.

*Symptoms.*—These are abrupt when due to perforation of gastric ulcer and are as follows: severe pain; vomiting of bilious or bloody material; embarrassment of respiration; subsequently chills, fever, and emaciation. Leukoyctosis and increased polynuclears during the suppurative period.

**Physical signs** depend on the quantity of air in the cavity and upon the presence or absence of a complicating pleurisy.

*Physical Signs (with Little Air Present).*—Dulness or flatness in the lower part of the thorax, but cough and expectoration are absent; signs of pressure in the pleural cavity are absent or slight, the thorax not being much dilated, and there being scarcely any obliteration of the intercostal spaces; the lungs are intact and distensible, and on deep inspiration there is vesicular breathing.

*Physical Signs (with Much Air Present).*—The lower part of the thorax protrudes and respiratory movements diminish; the heart is sometimes pushed upward and slightly to the right; the *liver extends well down into the abdomen*, occasionally as far as the umbilicus; the liver dulness in the back and lower part of the lung is replaced by a tympanitic zone; on auscultation the respiratory sounds are absent in this zone, and there are succussion sounds of a metallic pitch.

When pleurisy complicates subphrenic abscess from ulcer, there are the signs of pleurisy. Senator gives the following diagnostic points:

Violent pain in the epigastric and hypochondriac region; pain in the back on sitting up; pain on belching; patients prefer dorsal position when abscess complicates; while with pleurisy alone, they lie on the diseased side; edema of the lower lateral and posterior thoracic walls.

Pfuhl suggests a diagnostic point between *subphrenic abscess* and *pyopneumothorax.*

With subphrenic abscess, if an aspirating needle be inserted and a manometer be attached, the pressure is greater on inspiration and less on expiration.

With pyopneumothorax the pressure conditions are reversed.

Exploratory puncture is the accurate method of diagnosis, pus and food particles being aspirated.

**Terminations of Subphrenic Abscess.**—Perforation of the diaphragm and pleura; perforation of the lung, with expectoration of pus; or of the pericardium; or of the left ventricle; or of the colon; rarely perforation into the general peritoneum; or perforation of the skin, with resulting fistula.

Successful operations have been performed for subdiaphragmatic abscess, notably by Carl Beck.[1] Tuberculosis may occur in association with ulcer.

Some authors describe so-called atypic forms of ulcer, taking the most prominent symptom as a basis, such as: Gastralgic; catarrhal or vomiting; dyspeptic or latent; hemorrhagic; cachectic or perforative.

**Diagnosis.**—This is easy in the typic form characterized by hematemesis, the epigastric pain circumscribed and present during the digestive process, the dorsal pain, local tenderness, and vomiting. This constitutes the acute type of ulcer.

Cohnheim holds that real pain referred to the stomach is usually due to an organic lesion somewhere, and almost never to a neurosis. There are two methods of determining whether the pain is of gastric origin or not, and which may assist us in our diagnosis of gastric ulcer: First, gastric sedatives during the occurrence of the pain are given on an empty stomach, such as orthoform or anesthesin, 5 grains (0.3); or cocain, $\frac{1}{8}$ grain; or chloroform-water, 1 to 2 drams (4.0–8.0). If the pain is controlled directly, it is gastric (usually ulcer). The second method was devised by Bönninger.[2] After washing out the stomach and examining the washings for blood-cells, etc., he introduces through the tube 200 c.c. of a decinormal solution of hydrochloric acid (concentrated HCl, 3.6 c.c.; water, 1000 c.c.). If a gastric ulcer is present, this acid solution will probably cause immediate pain. If the pain is due to other causes, the test will be negative. This method is also used to show the progress and result of treatment. The test with orthoform or anesthesin may be tried.

In the less severe and non-typic cases (*i. e.*, without hematemesis) one frequently finds hyperchlorhydria, the presence of occult blood in the vomitus or stool, and pus on microscopic examination, either after the test-meal or on aspirating the empty stomach. With cases belonging to the acute type I have never failed to find occult blood, pus, or the combination, though sometimes several examinations may be necessary. The Mayos report blood as found in only about 33 per cent. of their gastric and duodenal ulcer cases. It will be noted that a large percentage of their operative cases were those in which food remnants (motor disturbances) were present. Cases with motor disturbances pointing to stenosis or spasm of the pylorus as the cause are, in the majority of instances, due to ulcer (benign or malignant) at the pylorus, and the condition is frequently chronic. In these cases one may frequently find pus in the gastric contents and occult blood more rarely, but the condition is surgical in any event. On the other hand, with chronic ulcer involving the body or curvatures of the stomach, and not lying within the pylorus, *pus is always found* according to my experience. Occult blood is sometimes present. *Pus* or *pus and blood* in the gastric contents, when not *ingested*, or *when there is no abscess* of the stomach, *show ulceration.* This was demonstrated in Connors' case already referred to. It is chiefly in the chronic cases that acidity is reduced. One must then determine whether the ulcer is benign or malignant by careful investigation into the history and observations as to rapid loss of weight, etc.

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[1] Medical Record, Feb. 5, 1896.

[2] Amer. Jour. Med. Sci., June, 1908.

**X-ray Diagnosis.**—The Röntgen rays have been employed as an aid to diagnosis. The method is based on the principle that a bismuth deposit occurs on the ulcerated surface and remains there for a period, even though the rest of the organ is clear. Adler[1] determines the motility of the stomach. He administers 1½ drams of bismuth subcarbonate in half a glass of water on an empty stomach. The picture is taken four to six hours later. This allows ample time for all bismuth to disappear from the stomach or duodenum, so that there alone remains that which is deposited in the *crater of an ulcer.* After this first *x*-ray examination, one ounce of bismuth subcarbonate is given in a glass of water, and a second picture taken immediately. The bismuth spreads rapidly over the gastric mucous membrane. This last can be facilitated by having the patient turn from side to side several times. By this second examination the shadow of the entire stomach is shown, so that the shadow obtained on the first plate can be localized with reference to its relation to the stomach.

Interpretation of the results requires considerable technical knowledge, the expense is considerable, and the method beyond the reach of many practitioners. It may be confirmatory evidence in some cases, but is not to be depended on alone. Other methods are described under *X*-rays. The writer believes them usually unnecessary.

Other conditions have been mistaken for gastric ulcer: *Gastric crises of locomotor ataxia;* absence of patellar reflexes. Romberg symptom and the Argyll-Robertson pupil are diagnostic.

*Duodenal Ulcer.*—This is at times impossible to differentiate, if the ulcer extends through the pylorus. There is a history of long duration, and the symptoms occur in attacks. Pus absent in gastric contents. It occurs most frequently *in males;* is often latent; melena is frequent; pain and tenderness are often a little more to the right than in gastric ulcer; no local dorsal pain spot; hematemesis not as frequent; pain after the ingestion of food is later than in gastric ulcer.

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[1] Jour. Amer. Med. Assoc., Nov. 12, 1910.

## DIFFERENTIAL DIAGNOSIS

| | Ulcer of stomach. | Nervous gastralgia. | Cancer. | Hyperchlorhydria. | Cholelithiasis. |
|---|---|---|---|---|---|
| Age. | Chiefly twenty to forty to fifty. | Eighteen to forty years. | Middle age and advanced life. | All periods, except in youth. | Over forty mostly; rare under twenty-five. |
| Sex. | More in women. | More in women. | More in men. | More in men. | Mostly in women. |
| Epigastric pain. | Intense; appears shortly after meals; increases on pressure; disappears at end of digestion; some free periods during the day at times, if stomach is empty. | Irregular; does not depend on food; relieved by pressure; duration of several days free period from pain. Disappearance of pain after electricity (Leube). | Pain less intense, but continuous; seldom free period, and is more steady; local tenderness. | Pain one to three hours after meals, and disappears after albuminous food soda bicarbonate are given. | Paroxysms of pain independent of eating often late and in the right hypochondrium. Pain over gall-bladder, which at times is enlarged to percussion and painful. Alkalis and albuminous food do not relieve pain. Deaver[1] states that in many cases the only symptom during a long period of time may be a sense of tightness or constriction in the epigastric region, and that this is rather a characteristic feature of gall-stone cases. The feeling may be also of stiffness or soreness on the right side. A sensation of chilliness may accompany it. A capricious appetite, constipation, flatulence, independent of the taking of food, and often discomfort on an empty stomach are among the early symptoms of gall-stone dyspepsia. Abrahams[2] holds that there is a *diagnostic sign* in *both acute and chronic cholelithiasis*, namely: Place the patient in the dorsal position, with arms and legs |

| | | | | | |
|---|---|---|---|---|---|
| | | | | | extended. Mark on the abdomen a point midway between the umbilicus and ninth right costal cartilage. A sudden thrust into this point invariably produces pain. |
| Dorsal pain. | Present. | Absent. | Absent. | Absent. | Head's gall-bladder zone is important. |
| Appetite. | Not impaired; afraid to eat. | Variable, peculiar cravings. | Poor. | Often increased. | Pain attacks patient in perfect health and radiates to right side or to right shoulder. It may cause collapse on account of severity. |
| Tongue. | Dry, red, and clean; or moist and smooth with light fur. | Normal. | Coated thickly. | Clean or slight fur. | Temperature and chills may occur. Liver may be enlarged and sensitive. Icterus is occasionally present. |
| Taste. | No abnormality. | No abnormality. | Bitter or sour. | No abnormality. | Gall-stones in stool in some cases. |
| Belching. | Absent usually; no odor if present. | Some. | Present, with disagreeable or fetid odor. | Absent. | Vomiting may be present. No dorsal pain. |
| | | | | | Hyperchlorhydria may be associated, but the history helps differentiation. Leukocytosis and increase in the polynuclears are present if there is acute inflammation, and aid in diagnosis. |
| | | | | | Muscular rigidity of upper quadrant of right rectus at times may be present.[3] · Rosin's test for biliverdin in the urine may aid diagnosis.[4] |

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[1] Early Symptoms of Upper Abdominal Disease, Jour. Amer. Med. Assoc., Jan. 20, 1910.

[2] New York Med. Jour., Jan. 8, 1910.

[3] Roth, Med. Record, April 23, 1910.

[4] Adler, Med. Record, Oct. 9, 1909, p. 599.

## DIFFERENTIAL DIAGNOSIS

| | Ulcer. | Nervous gastralgia. | Cancer. | Hyperchlorhydria. | Cholelithiasis. |
|---|---|---|---|---|---|
| Regurgitation. | Present at times; waterbrash with pyrosis. | Rare. | No waterbrash, pyrosis present. | Waterbrash and pyrosis. | |
| Vomiting. | Soon after meals, during digestion. | No regularity; occasional. | Once or twice a day, or every day or so, and not after each meal; quantity large. | None, unless hypersecretion be associated. | |
| Hematemesis. | Vomiting of often large amount; red or coffee ground; blood in stool (melena); often repeated same day or next day, and then none for some time. Occult blood usually present. | None. | Vomiting of blood, generally in small quantities; color usually of coffee grounds. May occur at frequent intervals. Is often decomposed and fetid in odor; melena. Occult blood at first. | None. | No occult blood in gastric contents, as a rule.[1] |
| Pus. | Present at some period in gastric contents. | No pus. | Pus present at some period in gastric contents. | No pus. | No pus in gastric contents. |
| Tumor. | None; unless thickened ulcer at pylorus, which then is smooth. | None. | Palpable; uneven; hard, tender, and readily movable. | None. | |
| Secretory function. | Increased, as a rule. HCl increased usually. Lactic acid absent. Chronic cases no increase. | Variable. No lactic acid. | HCl diminished or absent usually. Lactic acid present. | HCl increased. No lactic acid. | |
| Perforation. | May take place early. | Absent. | Rare; in last stages. | Absent. | |
| Complexion. | Often fresh; may be anemic after losses of blood; face anxious and suffering in old cases. Some loss of weight in old cases, but more gradual. | Pale. | Sallow and yellow; skin dry; peculiar and marked cachexia; rapid loss of weight. Leukocytosis is moderate; eosinophilia. | Often pale. | |
| Stool. | Occult blood often present. | None. | Occult blood often present. | None. | No occult blood in stool. |

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[1] This rule holds good when there is hyperchlorhydria. With achlorhydria hæmorrhagica gastrica, however, in association there are absence of free hydrochloric acid, mucus, many actively growing bacteria, pus, and occult blood. These gastric findings, the Mayos have shown, are often due to *gall-bladder disease* or *appendicitis* (Pilcher, Jour. Amer. Med. Assoc., Nov. 19, 1910).

Hunger pain, occurring two hours or more after the meal, is relieved by food. Occult blood is most frequent in the stool. (Under Differential Diagnosis of Duodenal Ulcer further data are given.)

*Spider Gall-bladder Adhesions.*—Robert T. Morris has demonstrated that gastric hemorrhage occurs at times with this condition. The stomach is dilated, and the diagnosis has been made of stenosis of the pylorus with ulcer.

There is a previous history of gall-bladder disease in these cases. The possibility of this condition must be considered.

*Cirrhosis of the Liver.*—Severe gastric hemorrhage may occur, but examination of the liver, the history, and other symptoms will differentiate.

**Location of the Ulcer.**—Occasionally one can make a probable diagnosis as to the position of the ulcer; if relief is afforded when standing, the ulcer is probably on the lesser curvature; if pain is intense on standing, it is on the greater curvature; if less severe pain when lying on left side, the ulcer is probably at the pylorus, etc. The position which affords most comfort to the patient is the *one which permits the ulcer to remain above the gastric contents.*

Einhorn[1] has recently described two methods for *recognizing* and localizing gastric ulcers: the so-called "thread impregnation test," in which the patient swallows the duodenal bucket with thread attachment, which is removed ten to twelve hours later. Blood discoloration on the string shows the presence of ulcer, and the distance of this spot from the teeth, its location. Sufficient blood to discolor the thread would certainly respond to the test for occult blood in the gastric contents. His second method, by the "gastric stamper," consists in the introduction of a collapsed balloon into the stomach, which is then distended, and secures an impression of blood from the ulcer. It is then deflated and removed. Both methods are uncertain, and pressure from inflation, I believe, dangerous. The use of the gastroscope to locate the ulcer is a risky procedure. The presence of blood or occult blood in the gastric contents, vomitus, or stool, the determination of pus, together with the methods of diagnosis previously described, are sufficient.

**Course.**—Gastric ulcer occasionally runs a rapid course, with death from perforation or hemorrhage. Stowell[2] states that 18 per cent. last a year or less; 46.5 per cent., from one to six years. A case of thirty years' duration has been reported. There are often intermissions of improvement and exacerbations; or the patient may become a chronic invalid.

**Prognosis.**—Excepting the fulminating cases, the more recent the ulcer, the more favorable the prognosis as to cure.

The mortality has been estimated at from 8 to 10 per cent.; some place it up to 20 per cent. In private practice Musser believes the mortality of ulcers treated medically to be 3.1 per cent., while in hospital cases it was 12.4 per cent. The position of the ulcer modifies the prognosis: if on the anterior wall, perforation is more apt to occur; if the pylorus is involved, stenosis and dilatation of the stomach result; if hypersecretion be associated, the results are less favorable. Stowell's statistics are as follows:

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[1] Med. Rec., April 3, 1909; Internat. Jour. of Surg., November, 1909; and Med. Rec., March 18, 1911.

[2] Med. Rec., July 8, 1905.

Death from hemorrhage, 3 to 4 per cent.; from exhaustion, 5 per cent.; from fatal perforation, 6.5 to 13 per cent. Pulmonary tuberculosis was the terminal event in 20 per cent. (Debove and Rémond) out of 100 cases investigated.

Greenough and Joslin, in the Massachusetts General Hospital, found that while 82 per cent. cases of ulcer were discharged as cured or relieved, only 40 per cent. remained well. Leube's marvelous statistics of medical cure, 90 per cent., are based on a very brief period and are of no value. Three years should elapse.

The statistics of 500 cases at the London hospital show that 50 per cent. were uncured by medical means, and of those discharged as cured, one-half relapsed. Parker Syms[1] believes that the mortality of cases treated medically will *reach beyond* 50 *per cent.*, since though the majority do not die from the immediate effects of the ulcer, yet the anemia and starvation so reduce their vital forces that they become a ready prey to intercurrent diseases. Eliminating the dangers of death from hemorrhage or perforation, the author believes the *acute type of ulcer* the most *favorable for cure*, while the *chronic cases*, as a rule, belong to the province of the surgeon. Lockwood[2] holds that the mortality for gastro-enterostomy for ulcer is 2 to 3 per cent. in the hands of a skilled surgeon, and 6 to 8 per cent. by the average surgeon; while in more complicated operations the mortality was 10 to 15 per cent. The writer believes that about 80 to 90 per cent. of chronic cases are cured by surgical intervention.

**Treatment.**—*For Hemorrhage* (*Hematemesis*).—Absolute rest in the dorsal position; immediate injection of morphin sulphate, $\frac{1}{8}$ to $\frac{1}{4}$ grain (0.008–0.016), and the application of a light ice-bag over the stomach, if necessary suspended from a barrel hoop to avoid weight. In emergency I have tied bits of ice in a sheet of rubber tissue or in dress shields.

One to two teaspoonfuls of a 5 to 10 per cent. solution of gelatin (cold), depending on the severity of the case, should be given by mouth every half-hour for ten to twelve hours, even if vomiting. This is an excellent hemostatic, and also takes up the free acid.

The frequent administration of small quantities is preferable to larger amounts given every two or three hours.

Gelatin, 3 ounces (100 c.c.) of a 2 per cent. solution by hypodermic with a large syringe, given between the lowest rib and crest of the ilium, is of value.

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[1] Some Surgical Aspects of Gastric Ulcer, N. Y. Med. Jour., July 16, 1910.

[2] The Prognosis and End Results of Treatment of Gastric Ulcer, Jour. Amer. Med. Assoc., April, 1911.

Ernutin (Burroughs, Wellcome & Co.), 5 to 10 minims (0.33–0.66), by hypodermic, or,

℞. Ext. ergot.................................. gr. xv (1.0);
Glycerini }
Aq. destil. } ................................āā ʒj (4.0).

by a large syringe subcutaneously have proved useful adjuncts; or fluidextract of ergot or hydrastis, 1 to 2 drams (4.0–8.0), in 2 ounces of water by rectum.

Chlorid or iron and acetate of lead are objectionable.

Tremolière has recently advocated a solution as a local styptic consisting of a 5 per cent. gelatin solution containing 2 per cent. chlorid of calcium. In a severe case I should not hesitate to give at once by mouth 1 to 2 ounces of a 10 per cent. gelatin solution containing 5 to 10 grains (0.3–0.6) of chlorid or, preferably, lactate of calcium.

The drug is dissolved in a little water and added to the gelatin solution while still warm, and then rapidly cooled off on ice.

The plain gelatin solution should be administered *in every case.*

Lactate, or chlorid of calcium, 10 to 15 grains (0.66–1.0), in 4 ounces (125 c.c.) of warm water can be given by enema as an adjuvant.

Lactate of strontium, or lactate of magnesium, 15 to 30 grains (1.0–2.0), in 2 to 3 ounces (125 to 200 c.c.) of sterile water, are recommended by hypodermoclysis, by Maas, to increase the coagulability of the blood, and would be valuable in gastric hemorrhage. Calcium chlorid should never be so given, as it causes local coagulation necrosis, and the lactate of calcium never stronger than 1:20 by hypodermic, and even so there is danger.

Adrenalin chlorid (1:1000), 5 to 15 minims (0.66–1.0), has been recommended by hypodermic for internal hemorrhage, but the pulse tension is markedly increased thereby, and I have seen secondary hemorrhage result. Five drops may be given by the mouth in 2 drams (8.0) of water for the local styptic effect if other remedies are insufficient.

Bismuth subnitrate, ½ dram (2.0) in 1 ounce of water, is at times of service.

Ewald recommends cautious lavage with ice-water after cocainizing the pharynx (2 per cent. solution of cocain is sufficiently strong by spray), in cases in which death seemed imminent from continuous hemorrhage. I have never found the procedure necessary, though I have treated severe cases. If it is employed, the addition of 4 ounces (125 c.c.) of a 5 per cent. gelatin solution, 15 grains (1.0) lactate of calcium, and 10 minims (0.66) adrenalin to the water for lavage would be of advantage.

J. Kaufmann[1] recommends gentle gastric lavage, the quantity of water at a washing about 300 c.c. The patient should lie down during the lavage, and the tube should be inserted just far enough to secure siphonage. If the hemorrhage has ceased, he does not wash the stomach, but only if the bleeding is continuing. He believes there is no danger of perforation unless the operator should overdistend the stomach by large quantities of water. He advocates the serum treatment of hemorrhage, and large doses of *crystalline bismuth subnitrate* to aid in the coagulation of the blood. Kaufmann further argues that the removal of the masses of clotted and disintegrated blood, which may cause gas formation and gastric distention, would lessen the chance of subsequent recurrence of the hemorrhage.

Wiel advises lavage with hot water at 42° C., but I doubt its advisability. W. L. Rodman advocates lavage with hot water at 120° to 130° F. When all *methods fail*, and *the hemorrhage continues or soon recurs*, opening the *stomach and direct suture of the bleeding ulcer has been advised.*

The administration of sterile horse-serum, as described by Hort shortly, might be of value. The hypodermic use of normal human blood-serum in cases of hemophilia neonatorum has been successfully employed by Welch.[2] It may be used for severe hematemesis in doses of 20 c.c. or more. Serum sickness, which might occur from the use of animal serum by this method, is thus avoided.

*For Thirst.*—The cold gelatin given for hemorrhage helps relieve thirst. A small piece of gauze dipped in cold water and held in the mouth, frequent washing of the latter; an occasional pellet of ice and enemata of normal saline solution at 105° F., 4 to 5 ounces (125 to 200 c.c.), if no relief by other methods, and given every three hours.

Proctoclysis, the administration of saline solution per rectum by the drop method, is also useful.

*Collapse.*—Caution must be employed not to overstimulate the heart, lest the coagulum be forced out and hemorrhage recur.

Only collapse that is fairly marked—a pulse 120 or over—should be treated, as depression of the pulse favors clotting. Among the valuable methods are:

External application of heat to the limbs by hot-water bags; rectal injections of 1 pint (500 c.c.) to 1 quart (liter) of hot normal salt solution at 115° to 120° F. every two to three hours; proctoclysis may also be employed.

Hypodermoclysis with normal saline solution, 6 ounces to 1 pint (375 to 500 c.c.), given at 105° F., through a large needle attached to a fountain syringe, preferably between the iliac crest and twelfth rib, is valuable, and does not stimulate too rapidly.

In emergency I saved one patient by employing an ordinary hypodermic needle and elevating the fountain syringe 6 to 8 feet to obtain forcible flow. In this event a higher temperature (118° F.)

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[1] Amer. Jour. Med. Sci., June, 1910.

[2] Ibid.

should be employed, as the fluid cools in passing through the small needle.

In one case, having no time to perform infusion, I needled a large superficial vein with a hypodermic needle[1] and infused by this method. Infusion with 1 liter of normal saline solution at 120° F. in severe cases is indicated. Direct infusion of blood from donor to patient is generally impractical, and the simple method is the best. Direct infusion from donor to donor may be employed in some cases.

Strychnin, $\frac{1}{60}$ to $\frac{1}{30}$ grain (0.00108–0.0021), every three or four hours by hypodermic, or pulverized camphor, 5 grains (0.3), in sterile almond oil, 20 minims (1.184 c.c.).

A single dose by hypodermic, and repeated in three or four hours, if necessary. Bandaging the extremities and elevation of the foot of the bed are of service.

Whisky or brandy, 1 to 2 ounces (30.0–60.0), may be added to the enema.

*Author's Method.*—Chief dependence should be placed on morphin, the ice-bag, gelatin, and lactate of calcium and bismuth subnitrate internally. Ernutin is useful and the serums may be employed if necessary.

The rational methods for the cure of acute or chronic gastric ulcer differ widely, the chief exponents being Leube-Ziemssen and Lenhartz.

*General Principles.*—There are certain *general principles* to which I must first refer.

Absolute rest in bed for a period of two to four weeks, even though the hemorrhage may not have taken place recently, gives the best results. The longest period is preferable.

Cruveilhier first recommended milk in gastric ulcer as an ideal food. Pure milk coagulates rapidly with some patients and does not agree. Boiled milk does not form large curds and leaves the stomach more rapidly. Lime-water and milk of magnesia lessen milk coagulability.

Lenhartz believes that the general nutrition should be improved as rapidly as possible to hasten the healing of the ulcer, and that sufficient milk to secure this result requires so large a quantity that it would overdistend the stomach. Though given in divided doses, he states that 2 to 3 quarts per diem are excessive, and limits the total quantity of milk to 1 liter, adding other materials to increase the calorie value.

Riegel demonstrated the capacity of egg-albumen to bind free hydrochloric acid, and also the use of sugar solutions to lessen acid secretion, such as pure dextrose or even ordinary dextrose. These possess high calorie value.

Soluble dry peptonoids, 1 ounce (30.0), have a calorie value of 120.4. They dissolve in milk or water. Milk powder, 100 grams;

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[1] Manual on Enteroclysis, Hypodermoclysis, and Infusion (Kemp).

Gärtner's fat milk; casein nutrose, 30 to 60 grams; somatose, 4 to 6 drams (16.0–24.0), and, notably, sanatogen, 20 to 30 grams per diem, possess nutritive qualities.

Among other foods are meat jelly, prepared by boiling chicken or beef with calves' feet (Fleming); Leube-Rosenthal's meat solution; flour soup boiled with milk, and barley-water or rice-water to dilute the milk.

Protection of the gastric mucosa and lessening hyperchlorhydria by the use of olive oil—½ to 1 ounce (16.0–32.0)—t. i. d. before meals, is of service.

Kaufmann[1] has demonstrated that the normal gastric mucus exercises a *protective influence* and is absent in ulcer, and that silver nitrate both aids the healing of ulcer and stimulates mucous secretion. Türck substantiates this by showing the increase of mycogen cells during the process of healing.

Large doses of bismuth act as a protective layer to the ulcer and prevent irritation.

Neutralization of the free hydrochloric acid is important. Among the most valuable alkalis are magnesia usta, milk of magnesia (Phillips), and soda bicarbonate. Magnesia preparations are also of use for the constipation. If their action becomes excessive, bismuth can be combined.

At times I employ soda bicarbonate in combination with magnesia. Bicarbonate of soda alone is apt to produce too much carbonic acid gas and distend the stomach.

In cases without dilatation, Carlsbad water or their salts are of service, as they lessen gastric secretion and empty the bowels. They form a permanent feature in Leube's cure.

Belladonna is valuable when there is hypersecretion, ⅓ grain (0.022) of the extract or 10 minims (0.6) of the tincture three times a day.

Iron and arsenic are imperative to improve the condition of the blood. Alcoholic drinks, coffee and tobacco, heavy salads, hot breads, acids, pastry, etc., should be excluded during the course of treatment.

If nutritive enemata are given for twenty-four hours following Leube's method, plain milk should never be employed, but should always be *peptonized.* This feature is often overlooked. An enema of saline solution should be administered an hour or more before the nutritive enema, in order to cleanse the bowel. The following is of service: Peptonized milk, 4 ounces (125 c.c.); raw egg, or whites of 2 eggs beaten up, with salt, 15 grains (1.00); sanatogen, 2 drams (8.0); water, q. s. 8 ounces (250 c.c.). It may be necessary to use a smaller enema, only 4 ounces (125 c.c.).

Somatose, 2 drams (8.0), or dry peptonoids, 1 dram (4.0), or liquid peptonoids, 1 ounce (32.0), may be substituted for sanatogen. Four nutritive enemata should be given during the day of sixteen hours.

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[1] Amer. Jour. Med. Sci., February, 1908.

Metzger has shown that wine in the enema increases gastric secretion.

Having enunciated the general principles of treatment, I will describe the chief methods employed and the procedures with which I have been most successful in my own experience.

Riegel confines himself exclusively to rectal feeding for six or eight days at the commencement of treatment following hematemesis, giving only a few pieces of ice by mouth, and then follows with a mild, non-irritating diet and the use of Carlsbad water or salts; while others confine the feeding to the rectum for two to three weeks.

Good results have been reported, but, as the patient is suffering from subnutrition, these methods can be improved upon.

*Leube-Ziemssen Rest Cure.*—This is substantially as follows: The patient is kept in bed for two to three weeks, not being allowed to rise for any purpose, either for defecation or urination; rectal feeding *for three days* if hematemesis has just occurred; hot poultices (flaxseed) over the stomach by day and warm Priessnitz compresses at night. Then after the first three days the subsequent diet for ten days should consist chiefly of milk, boiled milk, or milk with barley-water, strained barley, oatmeal- or rice-water, tea, and a little bouillon or meat extract. Unsweetened biscuits may be added. On the fourth day, for example, the patient receives 2½ pints of milk, 6 ounces of rusks (softened), and some meat extract. For the next ten days boiled calves' brain, boiled thymus, rice, and sago in milk, gruels and mushes, raw and soft-boiled eggs. This is followed by a little scraped rare or raw beef. Scraped raw ham and mashed potato for a week or so are added, and, finally, broiled chicken, venison, partridge, macaroni, white bread, roast beef, pike, and shad, etc.; coarse bread, skin, tendons, fruits, alcohol, and acids should be avoided.

Carlsbad water, a glass, or 1 to 2 drams (4.0–8.0) Carlsbad salts in 8 ounces (250 c.c.) of water should be taken half an hour before breakfast. This can be begun after the first week or ten days.

Ewald[1] uses nutrient enemas for three days after a hemorrhage, and then gives milk, butter, and eggs, and gradually increases the diet. He objects to Lenhartz's method, but practically employs a mixed method with not as satisfactory results.

Einhorn[2] employs nutritive enemata for a day or so after hematemesis, and then milk as the basis of his diet for the first two weeks; for the first week giving 5 ounces (150 c.c.) every hour, adding barley-water and bouillon; and gradually increasing the quantities of milk, but giving it at longer periods, adding eggs, crackers, etc.; at the end of ten days gradually increasing the diet. Recently he employs raw eggs and milk from the first day.

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[1] Centr. f. d. ges. Therap., Sept., 1906; also Deutsch. med. Woch., 1908, xxiv, 361.

[2] N. Y. Med. Jour., Nov. 20, 1909.

*Lenhartz's Method.*—Lenhartz[1] believes that many cases of gastric ulcer do not definitely improve, or but very slowly, under the method of entrenched milk feeding; that the high acidity is not measurably lessened; and that if patients are in a poor physical condition consequent upon one or more hemorrhages, often, indeed, in collapse, the "starvation treatment"—the ice and nutrient enemata and insufficient milk feeding following—not only maintain the patient in his anemic state, but may even drag him into serious inanition, and such an undermined constitution hardly favors the speedy healing of an ulcer. Frequent nutrient enemata excite the gastro-intestinal tract into peristaltic activity and may thus induce renewed bleeding; besides, very little nutriment is, after all, obtainable.

Should more milk be given by mouth, merely enough to preserve the body weight—3 liters for an adult—it would overfill the stomach and stretch its walls, thus preventing a contraction of the ulcer and again offering the danger of renewed bleeding. He advises another dietary treatment, one that will especially combat the hyperchorhydria and reinforce the enfeebled and anemic state of the patient.

The concentrated egg-albumen diet was tried. In case after case the effect proved so gratifying that this method became the routine treatment. The sour regurgitation subsides, the vomiting immediately ceases, the pain and distress after eating, within a few hours to a few days, disappear, and finally an increase in the body weight is manifest as early as the first week. Besides, the improvement is comparatively rapid, so that the patient can be dismissed as cured within a briefer time than formerly.

The following is the tabulated regimen: "Absolute rest in bed for at least four weeks. All mental excitement to be avoided. An ice-bag is placed upon the stomach and kept there almost continually for two weeks. This prevents gaseous distention and promotes contraction of the stomach walls, thus tending to obviate hemorrhage, and eases the pain when present. On the first day, even when a hematemesis has occurred, the patient receives between 6–9 ounces (200 and 300 c.c.) of iced milk given in spoonfuls, and from 2 to 4 beaten raw eggs within the first twenty-four hours. At the same time bismuth subnitrate is given twice or thrice a day, 30 grains (2 grams) per dose, and continued for ten days. The eggs are beaten up entire (with a little sugar), and the cup containing them is placed in a dish filled with ice, so that they remain cold. This food at once 'binds' the supersecreted acid, and, therefore, mitigates the pain rapidly and causes the vomiting, often quite troublesome, to cease. The fat which is present in the egg yolk also inhibits the secretion of hydrochloric acid. The portion of milk is increased daily per 3 ounces

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[1] International Congress at Wiesbaden, 1901; Therap. Gaz., Nov. 16, 1906; Mitt. a. d. Hamb. Staatsk., 1906, vi, 345.

(100 c.c.), and at the same time 1 additional egg is given, so that at the end of the first week the patient is receiving 25 ounces (800 c.c.) of milk and from 6 to 8 eggs. Both these foods are now continued in the same amount pro die for another week. No more than 1 *liter of milk a day* is allowed at any time. Besides milk and eggs, some raw chopped meat is given from the fourth to the eighth day, usually on the sixth, 9 drams (35 grams) pro die, in small divided doses (stirred up with the eggs or given alone); the day after 18 drams (70 grams), and later possibly more if well digested. The patient is now able to take some rice, well cooked, and a few zwieback (softened). In the third week quite a mixed diet is tolerated, the meat being given now well cooked or lightly broiled."

All heavy foods are interdicted, as well as vegetables with husks, etc., and those tending to produce flatulence. The patient is given strict orders to masticate his food thoroughly. This table gives the daily quantities.

The bowels are not moved, both in order to avoid any peristaltic irritation and to permit the reabsorption of blood that may have passed into the intestine. One need pay absolutely no attention to constipation in the first week, even in many cases to the end of the second. After the second week the bowels are moved with small glycerin injections or warm water, and after the third week this is done daily if a movement does not occur spontaneously. After this

| | Days after last hematemesis. | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 |
| Eggs[1] | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 8 | 8 | 8 | 8 | 8 | 8 | 8 |
| Sugar with eggs (gm.) | 0 | 0 | 20 | 20 | 30 | 30 | 40 | 40 | 50 | 50 | 50 | 50 | 50 | 50 |
| Milk (cc.) | 200 | 300 | 400 | 500 | 600 | 700 | 800 | 900 | 1000 | 1000 | 1000 | 1000 | 1000 | 1000 |
| Raw chopped meat (gm.) | 0 | 0 | 0 | 0 | 0 | 35 | 70 | 70 | 70 | 70 | 70 | 70 | 70 | 70 |
| Milk, rice (gm.) | 0 | 0 | 0 | 0 | 0 | 0 | 100 | 100 | 200 | 200 | 300 | 300 | 300 | 300 |
| Zwieback (gm.) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 20-<br>1 piece | 2 pieces | 2 | 3 | 3 | 4 | 5 |
| Raw ham (gm.) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 50 | 50 | 50 | 50 | 50 |
| Butter (gm.) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 20 | 40 | 40 | 40 | 40 |
| Calories | 280 | 420 | 637 | 777 | 966 | 1135 | 1588 | 1721 | 2138 | 2478 | 2941 | 2941 | 3007 | 3073 |

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[1] From the first to the seventh day inclusive the eggs are beaten; from the seventh to the fourteenth day inclusive one-half are beaten and one-half are cooked.

one tries to control the bowels by means of the food and by getting the patient to go to stool regularly.

For the anemia iron is given in the form of a soft preparation of Blaud's pills:

| | | |
|---|---|---|
| ℞. | Ferri sulphas | 10.0 gm.; |
| | Magnesia usta | 1.75 gm.; |
| | Glycerinum | gtt. xxx (3.6 gm.).—M. |

Divide in pilulæ lx; 2 pills to be taken two or three times a day.

The pills are given as early as the sixth, eighth, or tenth day of treatment, according to need, administering them first in a macerated condition.

In severe cases arsenic is also given in the form of "Asiatic pills," each containing 0.0001 gm. of arsenous acid. The dose is gradually increased, 3 for three days, 4 for four days, up to 7 for seven days, then decreasing again, 6 for six days, etc. After the tenth day and to the sixth week bismuth compositum is substituted for the subnitrate and given three times a day before meals. The patient is usually allowed up on the twenty-eighth day and is dismissed in the sixth to the tenth week. Lenhartz reports only 8 per cent. of recurrent hemorrhages after this method of treatment as compared with 20 per cent. after the older methods. No unfavorable results were produced.

Samuel Lambert,[1] of New York, and E. I. Spriggs,[2] of London, have reported favorable results.

*Senator's Method.*—Senator has modified Lenhartz's treatment, and employs a nourishing diet, non-irritating, which tends to check hemorrhage. It consists chiefly at first of gelatin, fat, and sugar. At first a 10 per cent. sweetened gelatin solution is given in tablespoonful doses every fifteen minutes to two hours. Small amounts of fresh butter and cream are allowed, the butter given in small frozen balls and the cream beaten up with sugar to form whipped cream, or it is iced. He gives daily 15 to 20 grams of gelatin, $\frac{1}{2}$ pint of cream, and 30 grams of butter. The daily allowance is from 900 to 1000 calories, and may be begun immediately after a hemorrhage. Olive oil may be given instead of butter. Gelatin is later replaced by calves'-foot jelly, milk and eggs, etc. He occasionally adds rectal feeding, though little of late. Gradual additions are made to the diet.

*Schmidt*[3] believes in the Lenhartz principle of feeding, but does not increase the diet as rapidly. He gives the stomach rest a few days, like Leube, and then increases the diet more rapidly by giving gelatin, eggs, butter, cream, sugar, and rice; but chopped meat and ham with caution.

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[1] Trans. Assoc. Amer. Phys.

[2] British Med. Jour., Apr. 3, 1909; Med. Chir. Trans., 1907, xc, 783; Proc. Roy. Soc. Med., 1909, ii, iii; Therap. Sect., 81; and British Med. Jour., May 21, 1910.

[3] Deutsch. med. Woch., Jan. 18, 1906.

E. Weiss, of Paris, has demonstrated the value of the injection of fresh animal serum in the treatment of hemophilia and purpura, and Wm. Hanna Thomson[1] has employed with success the injection of 15 c.c. of rabbit's serum hypodermically every day in a case of subcutaneous extravasation of blood and of hematuria. Hort[2] claims success in arresting severe hematemesis by administering normal horse-serum by mouth, and also excellent results in the *treatment of gastric ulcer without hemorrhage*, with the *subsidence of pain and vomiting*. He begins with 10 to 15 c.c. of sterile horse-serum, daily increasing it until no less than 30 c.c. are taken each day. The serum is administered by mouth in milk or in half an ounce to an ounce of water, and never on an empty stomach. If the pain is severe or there is hemorrhage, from 60 to 80 c.c. may be given in twenty-four hours. The treatment should continue for six weeks or longer, and preferably for some weeks after apparent cure. This *same treatment* is employed for *duodenal ulcer*. Hort employs serum treatment for chlorosis and externally locally for ulcers of the leg. Hort's dietetic treatment follows the lines of Lenhartz.

My own method depends upon whether I first treat the patient during the period of hemorrhage or later.

If the hemorrhage is taking place or has just occurred, 1 to 2 drams (4.0–8.0) of a 5 to 10 per cent. solution of sweetened gelatin is given cold every half hour for ten to twelve hours, even if there be vomiting. During the remaining period of the first twenty-four hours the gelatin is continued, ½ to 1 ounce (16.0–32.0), every two to three hours while the patient is awake, and in addition the whites of 2 raw eggs are beaten up and placed in a cup on ice and given in divided doses. An ice-bag is kept on for at least two weeks if hemorrhage has just occurred. The addition of horse-serum according to Hort's method the writer believes justifiable, or the use of human blood-serum by hypodermic.

*Gelatin treatment is continued for a week.* Scraped beef and ham are omitted for the first two weeks and cream substituted, to obtain the calorie values. A little sugar can be beaten up with the cream.

The day following the hemorrhage milk is begun, 6 ounces (200 c.c.) cold, in spoonful doses, 100 grams (10 per cent. gelatin solution) cold, in divided doses—½ ounce (16.0)—every three hours, and 1 raw egg beaten up and given in divided doses. These are placed in cups which are packed in ice. The milk is increased 100 c.c. daily up to 1000 c.c., and no more; eggs are increased daily 1 egg up to 8 a day. Sanatogen, preferably flavored, is begun on the third day (second day after hemorrhage). With the exceptions noted, the rest of the diet is after Lenhartz.

If no recent hemorrhage, I start the diet after the method corresponding to the third day in the table on page 278. An ice-bag is

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[1] N. Y. Med. Jour., June 11, 1910.

[2] Brit. Med. Jour., Jan. 5, 1910, p. 75; ibid., Oct. 10, 1909.

*Author's Modified Table.*

| First day, that of hemorrhage. | Days thereafter. | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 |
| Gelatin, ʒj to ij (4.0–8.0) of a 5 to 10 per cent. solution every half hour for ten to twelve hours. Then ℥ss to j (16.0–32.0) every two to three hours to total 200 to 300 gm ... | 100 | 100 | 100 | 100 | 100 | 100 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Eggs[1] (2 whites) on first day; entire thereafter | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 8 | 8 | 8 | 8 | 8 |
| Sugar with eggs, none.................... | 0 | 0 | 20 | 20 | 20 | 30 | 30 | 30 | 40 | 40 | 40 | 50 | 50 |
| Milk (cc.), none.......................... | 200 | 300 | 400 | 500 | 600 | 700 | 800 | 900 | 1000 | 1000 | 1000 | 1000 | 1000 |
| Cream (cc.), none........................ | 0 | 0 | 0 | 0 | 0 | 25 | 25 | 25 | 25 | 25 | 25 | 25 | 25 |
| Milk, rice (gm.), none.................... | 0 | 0 | 0 | 0 | 0 | 0 | 100 | 100 | 100 | 100 | 200 | 200 | 300 |
| Zwieback (gm.), none.................... | 0 | 0 | 0 | 0 | 0 | 0 | 20-<br>1 piece | 20 | 40 | 40 | 40 | 60 | 80<br>4 pieces |
| Sanatogen (gm.), none.................... | 0 | 10 | 10 | 10 | 20 | 20 | 20 | 20 | 20 | 20 | 20 | 20 | 20 |
| Butter (gm.), none...................... | 0 | 0 | 0 | 0 | 0 | 0 | 10 | 20 | 25 | 25 | 25 | 50 | 50 |
| Calories........................200 to 400 | 399 | 730 | 973 | 1272 | 1467 | 1765 | 2413 | 2476 | 2748 | 2822 | 3098 | 3349 | 3519 |

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[1] Eggs should be taken raw and well beaten for ten days, and after that 4 raw and 4 soft boiled.

kept on for two weeks if there is hemorrhage or one has occurred within a week or ten days.

Bismuth subnitrate, $\frac{1}{2}$–1 dram (2.0 to 4.0), is given in 2 ounces (60.0) water t. i. d. before feedings, commencing on the second day after hemorrhage. At times I combine magnesia usta, 15 grains (1.0), or sodium bicarbonate, 15 grains (1.0), with the bismuth.

*Pain.*—Tincture belladonna, 10 minims (0.66) in a teaspoonful of water should be given t. i. d. for pain. It also lessens gastric secretion and motility, and is a *valuable adjunct to the treatment.* At times a fourth dose may be indicated. It further lessens the tendency to vomiting and hemorrhage by diminution of the motility and of hypersecretion. Orthoform or anesthesin, of which the dose is 5 grains (0.3) is also recommended for the pain of ulcer. They are of some value, but as excessive acidity and increased motility have the most influence in these ulcer cases in aggravating the pain, belladonna seems of most value. Extract of belladonna, $\frac{1}{3}$ grain (0.22), or atropin, $\frac{1}{100}$ grain (0.0006), may be substituted for the tincture of belladonna.

Rarely a hypodermic of $\frac{1}{4}$ to $\frac{1}{2}$ grain (0.016–0.032) of codein or morphin, $\frac{1}{8}$ to $\frac{1}{4}$ grain (0.008–0.016), may be necessary for the acute pain.

*Bowels.*—I prefer to move the bowels gently by a small soapsuds enema containing 2 ounces (60.0) of olive oil on the third day after hemorrhage, and thereafter every other day. If no hemorrhage has occurred recently, then a daily movement should be secured. Milk of magnesia, 1 to 2 drams (4.0–8.0) in 4 ounces (125.0) water, given on rising, or small doses of Sprudel salts, 1 dram (4.0) in a glass of hot water, are of service.

On the seventh day after hemorrhage, or immediately if there has been no hemorrhage, iron and arsenic should be given:

℞. Blaud's iron pill........................ gr. v (0.3) (made fresh);
Sod. arsen............................. gr. $\frac{1}{50}$ (0.0013).

Pill made soft with honey and crumbled when taking, one pill t. i. d. after eating. The arsenic can be gradually increased to gr. $\frac{1}{25}$ (0.0026) sod. arsen. t. i. d.

I give no scraped beef until the commencement of the third week after hemorrhage, and then increase the diet after Leube's method. The bismuth treatment should be continued, or one can substitute the nitrate of silver treatment. It is advisable to keep this up for several months as a precaution, and then continue the diet and alkaline treatment for hyperchlorhydria for a considerable period.

At the end of three weeks the patient may be allowed to sit up for a short time daily; and at the end of four weeks to begin to go outdoors for a short time. In favorable cases it is advisable not to resume work under six weeks. Some of the chronic cases either will not remain in bed or cannot, for financial reasons.

In such event, we are obliged to employ careful but liberal feeding; the use of Carlsbad salts, iron and arsenic tonics, and either the subnitrate of bismuth treatment with an alkali in addition, or nitrate of silver. The following prescriptions are of service:

℞. Bismuth subnitrate, 2.0 to 4.0 (ʒss–j),
suspended in 4 ounces (125 c.c.) of water, and given t. i. d. half an hour before meals.

This is preferable to pouring the bismuth suspension through a stomach-tube.

As olive oil protects the surface and lessens the secretion, I sometimes employ the bismuth suspended in 1 to 2 ounces (30.0–60.0) of olive oil instead of water. Alkalis should also be used.

| | | |
|---|---|---|
| ℞. | Magnesia usta | 2.0 (gr. xxx) or |
| | Milk of magnesia | 4.0 to 8.0 (ʒj–ij). |

In 3 or 4 ounces (100–125 c.c.) of water alone, or combined with equal quantities of soda bicarbonate.

| | | |
|---|---|---|
| ℞. | Soda bicarb.,<br>Magnesia usta | āā 10.0 (ʒiiss); |
| | Milk-sugar | 2.0 (ʒss). |

Dose, 2.0 (ʒss) in water t. i. d. an hour after eating.

Nitrate of silver can be employed in place of the bismuth, to be given on an empty stomach t. i. d. half an hour before meals, as advocated by Boas and Kaufmann:

| | | |
|---|---|---|
| ℞. | Argenti nitratis | 0.2 (gr. iij); |
| | Aq. destil. | 180.0 (℥vj). |

Keep in dark bottle. Tablespoonful in wineglass of water t. i. d. half an hour before eating.

Occasionally lavage of the stomach with 1 : 5000 to 1 : 3000 silver nitrate has been advocated once or twice a week, if there has not been a hemorrhage for some weeks; but the internal administration is safer. It should be given for two to three weeks, then discontinued and the bismuth substituted.

The alkali can be given in addition t. i. d. an hour after meals. The meals should, preferably, be frequent and in smaller quantities, as in hyperchlorhydria.

In cases complicated by stenosis and ectasia lavage is necessary for the fermentation, and olive oil, 1 to 2 ounces (30.0–60.0) t. i. d. before the chief meals, to aid the passage of food. The application of Rose's belt, and the patient lying on the right side for half an hour after eating, both temporarily aid in emptying the stomach.

Resorcin in 5-grain (0.3) doses t. i. d. is also of value for the fermentation. Wm. H. Thomson[1] advocates the use of resorcin and also potassium bichromate in gastric ulcer. His formulae are found on page 201 under treatment of Chronic Catarrh of the Stomach. The writer prefers the omission of the nux and ginger in the ulcer cases.

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[1] N. Y. Med. Jour., June 11, 1910.

Hemorrhage is rare in this type, the ulcer being dormant and the chief symptoms due to stenosis. Surgery, gastro-enterostomy especially, is advocated in these cases. If there is tetany, the same procedure is advisable.

*Vomiting.*—In cases described by Lenhartz, this is relieved by neutralizing the acid, and this is the best treatment. Rarely, rectal feeding may be required for a few days. Bismuth subnitrate, 2 grains (0.13), and oxalate of cerium, 1 grain (0.065), should be given, or 1-drop doses of Fowler's solution of arsenic, four in all, an hour apart. If the vomiting continue, a single cautious lavage is a safer procedure than the risk of recurrent hemorrhage from the strain of emesis.

Tincture of belladonna, 10 minims (0.66), or atropin, $\frac{1}{100}$ grain (0.0060), are also of service for the vomiting.

*Pain from Adhesions.*—Rose's belt will at times aid in alleviating the pain due to the dragging of adhesions, by the support afforded to the viscera. Bacterial[1] vaccines have been employed.

*x-Rays.*—Bassler[2] advocates the employment of the *x*-rays for the healing of gastric ulcer, but I am skeptical of their value on the internal organs for such purpose. In cutaneous affections they are undoubtedly of value. Their prolonged use is also dangerous.

*Perforation.*—Temporarily, rectal feeding, a hypodermic of morphin, the ice-bag, and cautious lavage after cocainizing the pharynx, as suggested by Ewald, are indicated. *Immediate recourse should be had to laparotomy.*

*Surgery.*—The following are the indications for operation: Perforation with commencing peritonitis; local peritonitis, with or without abscess; subphrenic abscess; perigastric adhesions; ectasia due to stenosis from ulcer or spasm from its irritation; gastric tetany with ulcer.

In *recurring acute hemorrhages* the Mayos advise opening the stomach, locating the bleeding-point, suturing it firmly with catgut on the inner (mucous) side, and protecting the region by mattress sutures (musculoperitoneal). Gastrojejunostomy they have not found reliable.

In 6 cases—1 case of death from hemorrhage followed gastrojejunostomy—5 cases treated by primary operation on the bleeding point, with or without excision of the ulcer, recovered.[3]

Rovsing, in cases of severe hemorrhage, employs direct diaphanoscopy and gastroscopy (the light introduced through a minute incision in the stomach wall). After locating the source of hemorrhage, it is obliterated by running a suture about the bleeding surface. Kraft[4] reports 5 cases. I believe Mayo's method preferable.

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[1] On account of Türck's experiments in producing gastric ulcer in dogs by the ingestion of colon bacilli, a weekly dose of bacterial vaccines, 40,000,000 each, has been advocated by Aaron for the treatment of gastric ulcer.

[2] Diseases of the Stomach and Upper Alimentary Tract, Med. Times, Sept., 1909.

[3] Jour. Amer. Med. Assoc., Sept. 22, 1906.

[4] Archiv für Klinische Chir., Berlin, xciii, No. 3, pp. 557–789. Last indexed, Nov. 19, p. 1850. L. Kraft, Zur Behandlung der lebensgafährlichen Magenblutungen.

In cases of chronic ulcer, with recurrent hemorrhages, after a single systematic treatment for eight months to a year by the methods I have described, if the patient is still losing ground, I advocate resort to surgery. I am aware that some surgeons are loath to operate on such cases unless there is pyloric stenosis, and claim that in such event the gastric contents will not pass through the new opening, but through the pylorus. Canon and Blake have demonstrated this experimentally on animals. On the other hand, when a large anastomotic opening is made, this has been shown to be of practical value, especially by the posterior no-loop method.

It has been demonstrated that the pancreatic juice and bile entering the stomach aid in neutralizing the hyperacidity, and this is of service.

Wm. Mayo states that in chronic ulcer, where there is no mechanic obstruction, the result has not been as favorable. Unfortunately, we do find cases of multiple ulcers with hemorrhages from various areas and with an open and soft pylorus, such as Munro[1] reports, in which the results of surgery are not favorable; but in any event the lessening of hyperacidity through gastro-enterostomy is of value.

With chronic ulcer at the pylorus, pylorectomy, suture of the pyloric end, and gastro-enterostomy give the best results. The writer *now believes* that in the case of chronic gastric ulcer or ulcers (*extra pyloric*) which it may be inadvisable to excise, that, in *addition to the gastro-enterostomy*, infolding the stomach on the gastric side of the pylorus should be performed, thus closing the pyloric orifice. The *gastric contents must then of necessity pass through the gastro-enterostomy opening*. The same method would be useful in some cases of pyloric spasm.

Excision of the *ulcer in chronic cases*, when possible, after eight months' to a year's medical treatment without cure, I now *believe the safest procedure*. This is especially true in view of the *frequent occurrence of cancer engrafted on gastric ulcer*. Prophylaxis of gastric cancer can be aided by ulcer excision, as Wilson and MacCarty have shown that 71 per cent. of cancers had their origin in chronic gastric ulcer (Mayo).[2] Coffey[3] excised the entire lesser curvature in a case of ulcer with success. W. L. Rodman further advocates pylorectomy in chronic gastric ulcer to prevent later cancerous implantation on the base of the ulcer. Moreover, the danger of hemorrhage, adhesions, etc., are thus eliminated.

In cases of pyloric obstruction (stenosis) from ulcer with dilatation of the stomach, I always advocate surgical procedure, finding that though there may be temporary improvement at times quite marked under medical treatment, in every case there is subsequent relapse.

One must remember the possibility that after gastrojejunostomy for gastric ulcer a secondary ulcer occasionally occurs in the intestinal loop. A unique case of this type is reported by J. F. Percy, in the Journal of the American Medical Association, April 9, 1910, a recurrent ulcer perforating at the gastro-intestinal (anterior gastrojejunostomy) anastomoses. An omental adhesion prevented peritonitis. A posterior operation was then performed. Percy believes that infection from chronic cholecystitis is a factor in these cases. Dunham, in the November 10, 1910, issue of the same journal, gives some interesting statistics on gastro-enterostomy.

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[1] Annals of Surg., June, 1907.

[2] Jour. Amer. Med. Assoc., May 14, 1910, p. 1609.

[3] Ibid., Sept. 10, 1910 (Wilson and Willis); Jan. 28, 1911.

## EXULCERATIO SIMPLEX (DIEULAFOY) OR SUPERFICIAL ULCERATION OF THE STOMACH

Small ulcerations of the mucous membrane of the stomach, so minute that they appear to be little more than erosions, have been observed, from which fatal hemorrhage has taken place.

The usual symptoms of ulcer have been absent and no vomiting occurred prior to the hemorrhage. On the other hand, *symptoms of hyperacidity* may be present.

Dieulafoy was the first to minutely describe this affection.

**Anatomy.**—There is a superficial round or elliptic loss of substance, involving merely the mucous membrane and the muscularis mucosæ and some blood-vessels. It does not penetrate further into the coats of the organ. The defect may be from pinhead size to a quarter of a dollar in dimension; the margins are not indurated, and it may be difficult to detect even on autopsy, being concealed in some of the folds of the mucous membrane. The stomach is healthy throughout. There may be a slightly reddened zone about the area and it may be in any location.

**Etiology** is unknown. It may be the beginning of an ordinary ulcer, or possibly a toxic element is responsible.

*Age.*—Chiefly in persons from twenty-five to thirty.

**Symptoms.**—The patient may be in perfect health, when he suddenly vomits ½ liter (0.1) to 1 quart (liter) of blood, accompanied by melena. The hemorrhage may prove fatal. The symptoms are those of any hemorrhage: dizziness, cold extremities, collapse, and the patient may become rapidly moribund. Occasionally the patient may recover and suffer from recurrences. On the other hand, Dieulafoy holds that a case *representing severe symptoms of hyperacidity*, but *showing actual subacidity*, should always suggest exulceratio simplex ventriculi.

**Diagnosis.**—Ordinary cases of gastric ulcer suffer from gastric symptoms, while these cases may not.

From latent ulcer of the stomach it cannot be differentiated.

With chronic erosions there are no hemorrhages.

**Treatment.**—This is the same as in hematemesis from ulcer, morphin, ¼ grain (0.016); ice-bag over stomach; 10 per cent. gelatin solution every half hour, 2 drams (8.0) by mouth; lactate of calcium, 10 grains (0.66), by mouth and by rectum; ergot injections, hypodermoclysis (2 per cent. gelatin), also of saline solution, etc.

Dieulafoy recommends operative procedure, suturing the bleeding point if medical remedies fail or if the hemorrhages are recurrent.

## GASTRIC EROSIONS

These are subdivided into *acute erosions* and *chronic erosions.*

### Acute Erosions (Hemorrhagic Erosions)

These are small (2 to 4 mm.) abrasions of the mucosa of the stomach, in character usually multiple, and extend partly or through the layer. There is hemorrhage (hematemesis) with this type.

They occur in the newborn; in the cachexia of children; in chronic heart and arterial diseases; cirrhosis of the liver; in acute infections with the pneumococcus (Dieulafoy); and with septic organisms; also in postoperative cases, such as after appendicitis operation (vomito-negro-appendiculaire), and associated with the throbbing aorta in a considerable number of neurotic women.

**Treatment** is that of hematemesis and also of the cause.

### Chronic Erosions (Erosions of the Stomach)

An erosion is a small superficial exfoliation of the gastric mucous membrane. Erosions of the stomach have been quite frequently found at autopsy, and the subject has been discussed by Virchow, Ewald, Gerhart, and others. The latter found nothing characteristic.

Riegel notes the frequency with which small fragments of mucous membrane are washed out of the stomach, and believes it due to the tearing from this procedure. He denies its significance as a special pathologic process, but believes that at times an examination of the fragments will demonstrate the general condition of the mucous membrane.

There is even to-day some dispute as to whether the erosions of the stomach can be described as a separate clinical entity, in view of the fact that in the majority of cases there is a chronic gastritis, and only on lavage the bits of mucous membrane are secured, some believing it to be due to manipulation with the tube.

We must credit Einhorn[1] with first describing erosions of the stomach as a clinical entity. Pariser,[2] Quintard,[3] Mintz,[4] and others have reported cases.

Undoubtedly, not in every case in which bits of mucous membrane are washed out of the fasting stomach have we chronic erosions. I have noted in several cases of chronic gastritis—in which unquestionably lavage was performed in an unscientific manner by the physician in attendance—the appearance of these small fragments, and yet the patient never subsequently suffered from the salient symptoms described. Traumatism was the evident cause.

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[1] N. Y. Med. Rec., June 23, 1894; also Jour. Am. Med. Assoc., May 20, 1894.

[2] Berlin. klin. Wochenschr., 1900, No. 43.

[3] Arch. f. Verdauungskrankheiten, 1901.

[4] Zeitschr. f. klin. Med., Bd. 46, 1902.

Erosions may occur in acute cases. I was recently called to attend a patient with acute gastritis, having severe and persistent vomiting streaked with blood, and though I washed the stomach with greatest care, in two washings several pieces of gastric mucous membrane were found, evidently exfoliation from the acute process and violent vomiting. The case made a rapid recovery with no further symptoms.

Unrecognized cases of chronic erosions occur, but those recognized intravitam by the clinical symptoms are, so far reported, comparatively few.

**Etiology.**—Chronic catarrhal gastritis is the chief factor, though Einhorn reports erosions associated with hyperchlorhydria, and I have seen them once with acid gastritis.

**Symptoms.**—The diagnostic symptoms described by Einhorn are: Pain, emaciation, weakness and lassitude, and the finding in the wash-water after lavage of one or more small pieces of gastric mucous membrane. There is usually decrease in acidity and free hydrochloric acid and considerable mucus (chronic gastritis).

*Pains.*—These are not intense and occur directly after meals, irrespective of the character of the food. They last one or two hours and are never severe. There are usually intervals free from pain, though rarely the pains are constant.

*Emaciation.*—The patients lose weight at first; the face becomes rather thin. They have not the cachexia of cancer nor the appearance of suffering as in ulcer.

*Weakness.*—The patient feels weak and unable to work, and likes keeping quiet, most markedly so for a period after meals. Loss of appetite is present in some cases.

**Pathology.**—If the stomach is washed out in the fasting condition, one or more small pieces of gastric mucous membrane are found in the wash-water. They appear normal under the microscope, but are infiltrated with red blood-cells. This lesion is constantly found after lavage.

Blood is rarely present, though occasionally the wash-water is slightly streaked with it.

Einhorn believes the " erosions " resulting from the peeling off of the mucous membrane are responsible for the pain and tenderness, and that it has not yet been determined whether the exfoliation recurs at the same place after healing or in new regions of the stomach.

N. W. Jones[1] describes hemorrhagic erosions and autopsy findings in chronic erosions (Einhorn's disease). Considerable blood was found in the intestines, and blood had previously been found in the stools. The cause of death is not given. The case is apparently one of acute hemorrhagic erosions, recurrent type, or possibly more the aspects of achlorhydria hæmorrhagica gastrica, and not of chronic erosions at all.

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[1] Jour. Amer. Med. Assoc., Oct. 14, 1911.

*Gastric Analysis.*—The hydrochloric acid secretion is, as a rule, decreased and also the total acidity. Considerable mucus is usually found. Rarely, hyperchlorhydria is present, and in one case I found acid gastritis.

In washing the fasting stomach, one to four pieces of mucous membrane (0.3 to 0.4 cm.) are found. They are blood-red, and under the microscope the gastric glands and red corpuscles are visible.

**Prognosis.**—The disease is usually of long duration, with at times intervals of improvement.

**Treatment.**—The diet depends on the gastric findings. The treatment is the same as for chronic gastritis, acid gastritis, or for hyperchlorhydria, depending on the case. Hydrotherapy and out-of-door exercise are of service.

If deficient hydrochloric acid, nux vomica, compound tincture of cinchona, and condurango are indicated; if hyperacidity, the alkalis, such as magnesia usta or sodium bicarbonate.

There are three methods of local treatment:

1. *Bismuth Treatment.*—Lavage every other day with milk of magnesia to dissolve the mucus, and t. i. d. 15 to 30 grains (1.0–2.0) of bismuth subnitrate before meals.

2. *Nitrate of Silver Treatment.*—This is superior in many cases. It may be administered internally, $\frac{1}{4}$ grain (0.016) in solution t. i. d. three hours after eating; lavage with an alkali every two or three days depending on the mucus; or lavage every other day with 1 : 2000 to 1 : 1000 silver nitrate, preceding it by lavage with warm water.

Einhorn recommends intragastric galvanization on one day, with nitrate of silver spray preceded by lavage on the following day, and so alternating.

He first washes the stomach with warm water, which is all removed, and then sprays the stomach with his instrument, employing 10 c.c. of a 0.1 to 0.2 per cent. solution of nitrate of silver solution, employing most of it and moving the tube about. The bottle should be opened before removal of the spray.

3. The extract of the suprarenal gland (Armour & Co.) has been recommended by Einhorn. He employs it in his powder-blower and applies it every other day to the stomach, 3 grains (0.194) at a time, instead of the silver nitrate spray. Good reports are given of this method.

## PERIGASTRITIS AND PERIGASTRIC ADHESIONS

Localized perigastritis with the formation of adhesions to other organs may result from various causes, among which are the following: gastric ulcer; recurrent attacks of acute gastritis by extension or by infection from within; infections from the gall-bladder, liver, appendix, spleen, duodenum, or transverse colon; cancer or ulcer of adjacent organs; and tubercular peritonitis. Adhesions are more frequently due to gastric ulcer and are not at all uncommon in cases in which cicatrization has taken place, in which event the pylorus and lesser curvature are most frequently involved. In at least 7 to 8 per cent. of cases of gastric ulcer such adhesions occur. They may also take place to the parietal peritoneum. Infection of the gall-bladder is a quite frequent cause of gastric adhesions, particularly of the spider web variety, which may produce pyloric stenosis with dilatation of the stomach, and at times even gastric hemorrhage from the congestion caused by circulatory interference, so that the diagnosis of stenosing gastric ulcer has been made. R. T. Morris particularly refers to this type. The stomach, liver, and gall-bladder may be bound together by adhesions originating from either gastric ulcer or gall-bladder infection. When the adhesions result, for example, from gastric ulcer, there is at first a localized peritonitis (perigastritis) with increased pain, tenderness, at times vomiting, and a moderate temperature in the cases of more acute type. The location of the pain and tenderness depends on the site of the perigastritis. In the subacute cases there may be few clinical symptoms, possibly an increase in the gastralgia, with some interference in the motility of the stomach, increased pain when the stomach is distended with food, some loss of flesh and strength, etc. If ulcer be the cause, the gastric findings do not alter. Disturbances of gastric secretion, however, frequently occur with gall-bladder disease (infection) with accompanying adhesions. J. A. Lichty[1] has experimentally demonstrated on dogs disturbances of the gastric functions produced by gall-bladder infection. If the adhesions affect the pylorus, stenotic dilatation of the stomach may result with its accompanying symptoms. Gastroptosis may also occur from adhesions forming on the lower border of the stomach and dragging down the organ. Adhesions to the intestines may produce acute or chronic intestinal obstruction and distention of the gall-bladder from interference with the common duct. If adhesions involve the pancreas affecting the duct, disturbance of the pancreatic functions may result.

A local abscess may occasionally occur which may require surgical intervention. In some cases one can palpate a thickened mass due to the perigastritis.

If the perigastritis and adhesions occur at the cardia, partial stenosis may result, with regurgitation of food, etc., the symptoms of stenosis of the esophagus.

A frequent symptom observed is that when adhesions are present in the stomach, exclusive of the cardia, as above noted, after distension of the stomach with food or gas, considerable pain often follows from dragging on the adhesions.

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[1] Amer. Jour. Med. Sci., Jan., 1911.

**Diagnosis.**—The previous history of gastric ulcer or some other intra-abdominal lesion is of great assistance in the diagnosis. Local tenderness and pain, increased pain after distention of the stomach with food, the symptoms of pyloric stenosis with dilatation of the stomach (when such conditions are present) are of service. Sometimes a thickened mass can be palpated.

The *x*-rays are a valuable aid, showing local disturbance in the motility of the stomach, a change of contour, and in some cases an abnormal position of the organ. The reader is referred to the *X*-ray Diagnosis of Stomach Disease, page 108, where, for example, the abnormal position of the pylorus when adherent to the gall-bladder is well illustrated (Fig. 69).

**Treatment.**—Perigastric adhesions in most cases require operative interference. If a localized abscess is present with the perigastritis, it should be opened and drained. Mild types of adhesions may go for years without operation, but there is invariably considerable discomfort, and frequently disturbances of the gastric secretion ultimately result, as well as disturbances of motility of the organ. The nervous strain on the patient is marked and invalidism results. The author advocates operation by preference even on these cases.

Adhesions producing pyloric stenosis require operative procedure.

## CHAPTER XIII

# CANCER OF THE STOMACH (CARCINOMA VENTRICULI)—OTHER TUMORS OF THE STOMACH—APPARENT TUMORS OF THE STOMACH—FOREIGN BODIES IN THE STOMACH

## CANCER OF THE STOMACH (CARCINOMA VENTRICULI)

**Frequency.**—In an anlysis of 30,000 cases of cancer, W. H. Welch, of the Johns Hopkins Hospital, finds the stomach involved in 21.4 per cent., standing next in frequency to uterine cancer. Osler states there were 150 cases of carcinoma ventriculi in 8464 patients admitted to the hospital wards. Häberlin gives 41 per cent. from 1877 to 1886 in his statistics, while Brinton places it at about 25 per cent. Virchow stated that the stomach was the site of primary carcinoma in 34.9 per cent. of all cases, while d'Espine places it at 45 per cent. The Census Bureau reports out of a total of 140,088 deaths from cancer, the stomach and liver combined were 36.4 per cent., and the female genitals 14.7 per cent. In a study of 2268 autopsies at the Philadelphia General Hospital,[1] the total number of cases of primary cancer were 121. Of these, 45 were of the stomach; 32 were in males, 13 in females. There were 13 cases of uterine cancer. Küttner[2] comments on the prevalence of cancer in southeastern Germany. Häberlin demonstrated that gastric cancer is on the increase in Switzerland, and Joseph D. Bryant shows the same in the United States. Its frequency seems to vary in different countries, Griesinger having never observed it in Egypt. Coley[3] shows the general increase of cancer.

*Age.*—Welch finds that three-fourths of his cases occurred between the ages of forty and seventy. Osler analyzes 150 cases as follows:

Between twenty and thirty years, 6 cases; from thirty to forty, 17; forty to fifty, 38; fifty to sixty, 49; sixty to seventy, 36; seventy to eighty, 4.

Fifty-eight per cent. occurred between forty and sixty, the youngest case was twenty-two. Welch's statistics show the majority of cases between forty and sixty. The maximum liability to cancer of the stomach is, therefore, between the ages of forty and sixty.

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[1] Cancer of the Stomach, A Statistical Study (J. A. McGlinn), Amer. Jour. Surg., Dec., 1909.

[2] Therapie der Gegenwart, Berlin, Jan., 1911, No. 1.

[3] Surgery, Gynecology, and Obstetrics, June, 1910, pp. 591–597.

Two cases of congenital carcinoma have been reported and also a few cases under the age of thirty.

*Sex.*—Welch finds cancer of the stomach slightly more frequent among men, 1233 men to 981 women; and Osler, in 150 cases, 126 males and 24 females. Statistics vary somewhat regarding percentages, but it seems to preponderate in males.

*Race.*—Among 150 cases at the Johns Hopkins Hospital, there were 131 among the whites and 19 among the blacks. The ratio of colored patients to white, however, in our hospitals is small.

*Heredity.*—Since several members of one family have been afflicted with cancer, many are inclined to believe heredity plays a part. Delafield[1] and Prudden hold that, "while the influence of heredity is difficult to estimate, there are a few well authenticated cases of the remarkable prevalence of malignant tumors in families within a few generations."

The statistics of Williams showed that in 235 cases of carcinoma of the uterus or breast, 9 per cent. gave a history of carcinoma in the father or mother, while in nearly 20 per cent. there was evidence of carcinoma in the family; though such statistics are suggestive and indicate that an hereditary predisposition to the development of tumor may exist, this does not account for the immediate excitement of the growth of tumors, and is, as Menèstrier has urged, but one of the examples of hereditary disposition which is observed in many forms of disease, such as infections, cerebral apoplexy, etc.

Williams cites the fact that the father, brother, and two sisters of Napoleon died of cancer of the stomach, to which he himself succumbed.

William S. Bainbridge,[2] who has extensively investigated the question, believes that the hereditary acquirement of cancer requires much more study before definite conclusions can be formulated, and in this I agree.

**Etiology.**—Traumatism has been given as a frequent cause of cancer of the stomach, but Osler reports only 1 case in his series. Probably attention is drawn to this part by reason of the injury, and the tumor is thus recognized more early. Coley[3] has, however, recently reported a number of cases in his own experience, and in that of foreign observers, in which traumatism would seem to act as a direct causative agent, both of carcinoma and sarcoma. John B. Deaver[4] refers to the influence of trauma, citing as an example skin carcinoma caused by continued exposure to the *x*-ray. Cider, sour wines, mental worry, and nervous strain have been suggested as predisposing causes, but they have no influence.

As the muscular fibers of the cardia and pylorus, undergo frequent expansion and contraction, and are subject to more work than other portions of the stomach, Brinton believes the necessarily increased nutrition of these parts may favor glandular proliferation and be productive of a neoplasm.

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[1] Handbook of Pathologic Anatomy and Histology.

[2] Boston Med. and Surg. Jour., June 27, 1907.

[3] Annals of Surg., April and May, 1911.

[4] Amer. Jour. of Surg., Aug., 1911.

Some consider chronic inflammatory disease of the mucous membrane of the stomach to be a predisposing factor in the production of carcinoma, notably the polypoid form of chronic gastritis (Menestrier). As a rule, carcinoma develops without a previous history of long-standing gastric disturbance, and I agree with Ewald and Einhorn in believing these conditions have no influence.

The gastritis found with cancer is a secondary condition. The development of cancer on an ulcer scar has been clinically demonstrated by Hauser. Häberlin places about 7 per cent. of cases as occurring in this manner, while Moynihan gives 60 per cent., and William Mayo 36 per cent. It is now held that about 71 per cent.[1] of cases develop from ulcer.

When one remembers that in about 5 per cent. of persons dying from all causes Brinton finds evidences of gastric ulcer, it can be readily understood how *carcinoma can develop on an ulcer scar with no apparent previous gastric symptoms*. This is the author's view; 60 to 70 per cent. developed on a previous ulcer in spite of the fact that Fenwick reports only 3 per cent. of cases with previous ulcer history, and Osler 2.6 per cent. These last statistics do not militate against the theory of development of carcinoma on the base of an occult ulcer with no symptoms.

Regarding the parasitic origin of cancer and its infectious nature there is much dispute. Scheurlein believed he had discovered a bacillus, but later researches demonstrated his error.

Gaylord, Park, and Adami hold to the parasitic theory, and Gaylord states that in all the organs, including the blood taken from cases dying of cancer, certain organisms (parasites) are found. He has inoculated guinea-pigs and dogs with peritoneal fluid from a human abdominal tumor and produced adenocarcinoma in the lung and liver. Coley has secured, in a few cases, good results in the treatment of cases chiefly of sarcomata and of a few cases of carcinoma by the injection of his serum, and I have personally seen a favorable result in 1 case of kidney sarcoma.

Psorosperms have been found in cancer cells, but it has not yet been proved whether they are real psorosperms or dried-up and changed cells. Beard's recent theory and his suggestion of the use of trypsin in the treatment of cancer has been proved of *no value*. We must confess so far the origin of cancer has not yet been determined, though I believe it will eventually be demonstrated to be due to some bacillus or parasite.

**Morbid Anatomy.**—Waldeyer demonstrated that cancer of the stomach originated from the glandular structure of the mucous membrane, being an atypic proliferation of the epithelium of the glands. Beginning in the mucosa, it infiltrates the submucosa, the muscular coat, and extends to the serosa. Early in the development of the disease the lymphatic glands become enlarged, particularly those of the lesser curvature. Metastatic growths may take place. The cancerous growth, especially of a certain type, may slough and form irregular ulcers.

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[1] Wilson and MacCarty, Amer. Jour. Med. Sci., 1909, cxxxviii, p. 846.

**Varieties.**—The most common varieites of cancer are the cylindric-celled adenocarcinoma (or epithelioma) and the encephaloid or medullary carcinoma; next in frequency is the scirrhus, and least frequent, the colloid cancer.

1. *Adenocarcinoma* (*Cylindric-celled*) *or Epithelioma.*—This type forms soft tumors, of firmer consistency than the medullary type, and sloughing more slowly. Microscopically, the section shows elongated tubular spaces filled with columnar epithelium, and the intervening stroma is abundant. Gradually the tubular spaces develop into cell-nests. There is frequently infiltration of the connective tissue with white blood-corpuscles. Cystic degeneration is quite common. Metastases and hemorrhage may occur.

2. *Medullary Carcinoma.*—This occurs in soft, spongy, fungating masses, which involve all the coats of the stomach and usually ulcerate early. It is large and often flat, projecting above the mucous membrane, and may form villous-like projections, or a cauliflower-like outgrowth. It is soft and grayish or yellowish white and contains many blood-vessels and cells. Microscopically, it shows scanty stroma, enclosing alveoli containing irregular polyhedral and cylindric cells. It is often blackish in color, due to hemorrhage (melanotic), and has a tendency to ulcerate. Metastases are frequent.

3. *Scirrhous* (*Fibrous*) *Carcinoma.*—This is characterized by great hardness, due to abundance of stroma and the limited amount of alveolar structure. The large amount of connective tissue makes the tumor very firm and compact. It cuts almost like cartilage, and on section has a yellow or grayish-white appearance. There is little tendency to ulcerate, except at a late stage superficially, and secondary metastases are not common. It is seen quite frequently at the pylorus, there being a diffuse thickening and hardening of the wall and then a contraction, being a common cause of stenosis. The tumor may be diffuse, involving all parts of the stomach, when it may be difficult to recognize it microscopically from cirrhosis ventriculi. It has occurred in the stomach secondary to ovarian cancer, and as a part of a diffuse carcinomatosis, with involvement of the small and large intestines. It may be combined with the medullary form.

4. *Colloid Carcinoma.*—This type of cancer is peculiar, from the fact that it invades widely all the coats of the stomach. It spreads with great frequency to the neighboring parts, and at times causes secondary growths of the same nature in other organs.

The appearance on section is distinctive, showing large alveoli filled with translucent gelatinous colloid material. This is often present, even to the naked eye. On scraping, no cancer juice exudes, but gelatinous fragments.

Various transitional forms, from one variety to another, are often found. Rarely a carcinoma consisting of squamous epithelium may extend from the esophagus into the cardia.

Brinton, in analyzing 180 cases of cancer, finds the scirrhus type to be most common (72 per cent.), the medullary next in frequency, though other observers, notably Osler, consider the epithelioma to be most frequent.

Cancer of the stomach is usually primary, though secondary growths have been reported.

Cancer may also extend from the liver, pancreas, and intestines.

**Types of Growth.**—Medullary and colloid cancers involve large areas of mucous membrane, growing little above the surface, being somewhat flattened, with occasional rough nodulous masses. Blood extravasations and adhesions to neighboring organs are of frequent occurrence.

The scirrhus variety extends usually only over a small portion of the mucosa, and may develop extensively in thickness, growing in depth and height. The latter type, however, occasionally infiltrates the entire stomach, causing a contraction of the organ (cancer atrophicans).

**Secondary Changes in the Mucous Membrane of the Stomach.**—Hammerschlag has investigated the gastric mucosa in cases of carcinoma by examining fresh pieces of mucous membrane removed in cases of resection of the pylorus at the time of gastro-enterostomy, examining also the section of the stomach.

When the hydrochloric acid secretion was intact, there were no changes in the mucous membrane. When it was absent and lactic acid present, changes occurred, and there were found destruction of the rennet glands in certain areas and small-celled infiltration and formation of connective tissue in the gastric mucosa, also mucoid changes or cystic degeneration; in effect, a secondary atrophic gastritis. Eosinophile cells are present. Hypertrophy of the muscular fibers and connective tissue has been observed. Ewald has also noted that the entire mucosa may show the lesions of chronic gastritis. It is interesting to learn that Fenwick has demonstrated that atrophic gastritis may occur with carcinoma of the breast and uterus. Wilson and MacCarty, however, in an extensive study of 230 stomachs resected by William Mayo for carcinoma of the stomach, in the majority of which free hydrochloric acid was absent, found that in *none of them* was there a general atrophy of the mucous membrane. There was present, almost without exception, a marked proliferation of the mucous membrane at the margin of the ulcer, but usually an accompanying round-cell infiltration of the submucosa of a greater or less degree.

**Location of the Cancer.**—The development of the cancer may take place in various regions of the stomach, at the pylorus or cardia or within the organ, causing variation in the symptoms according to location, and in each case necessitating a special plan of treatment.

Welch's analysis of 1300 cases is as follows: Pyloric region, 791; lesser curvature, 148; cardia, 104; posterior wall, 68; the whole or greater part of the stomach, 61; multiple tumors, 45; greater curvature, 34; anterior wall, 38; fundus, 19.

In Brinton's cases, 60 per cent. were found at the pylorus. The latter is evidently the point of selection.

Wm. Mayo believes 70 per cent. involve the pyloric region, and that 60 per cent. originate in the pylorus or within 3 inches of it.

**Changes in the Shape of the Stomach.**—If the cancer is situated at the cardia, the stomach is usually retracted and small in size, while the esophagus above the stricture is dilated. If the tumor constricts the pylorus, the stomach will be dilated. Gastroptosis of varying degree may be present by reason of the weight of the tumor dragging down the pylorus, and it may even lie down in the pelvis. Adhesions may distort the shape of the organ, and Riegel reports an hour-glass contraction of the stomach resulting from cancer.

**Perforation.**—Perforations into other viscera, through the skin, or into the general peritoneal cavity are rare. The aorta has been perforated. Subphrenic abscess has been produced. Perforation, however, seldom occurs.

**Cancerous Metastases.**—In an analysis of 1574 cases by Welch, metastases occurred in the lymphatic glands in 551; in the liver, 475; in the peritoneum, omentum, and intestines, 357; in the pancreas, 122; in the pleura and lung, 98; in the spleen, 26; in the brain and meninges, 6; in other parts, 92.

Some interesting material has been furnished by Elsner[1] regarding *cancerous metastases of the nervous system* from cancer of the *stomach or intestines*. He describes peripheral invasion with symptoms of multiple neuritis. Klippel[2] reports 5 cases of this type. Schlesinger[3] gives statistics as to spinal invasion. Elsner reports 3 cases, in one of which spinal symptoms preceded the gastric symptoms, and in another case the spinal symptoms were so severe that they *masked the presence* of the primary stomach growth. The bodies of the vertebræ were first involved. These spinal cases are characterized by "agonizing pain." Pinatelle and Cavaillon[4] describe 2 cases of gastric cancer with secondary deposits in the cranial bones and meninges, and Holden[5] reports a case of invasion of the optic nerve secondary to gastric carcinoma.

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[1] N. Y. Med. Jour., Jan. 21, 1911.

[2] Thèse, Paris, 1889.

[3] Beiträge zur Klinik des Rückenmarks und Wirheltumoren, Jena, 1898.

[4] Progrès Médical, April 14, 1906.

[5] Arch. of Ophthalmol., 1902, xxi, pp. 427–432.

The abdominal lymph-glands are usually affected, but the *cervical and inguinal glands* are sometimes attacked.

Secondary growths may occur at the navel or in the skin in the immediate vicinity. Infection occurs either by the blood-vessels or lymph-channels. Direct extension by continuity may take place.

The medullary and colloid types of carcinoma are often associated with metastases. Sometimes numerous small cancerous deposits occur in the pleura. The microscope will differentiate them from tuberculosis, though they have occurred together. Rarely, metastases are found in the eyes, as noted above.

**Symptoms.**—There are general symptoms and special symptoms, depending on the location of the growth.

**General Symptoms.**—Usually a patient of middle age, fifty to sixty years, up to a short time previous—a few months or so—being in perfect health and having had no gastric symptoms, will begin to complain of slight dyspeptic disturbance, loss of appetite, and fulness, pressure, and discomfort after eating. The author believes that this statement *must be modified*, in view of the fact that a *careful history* will often elicit some previous *gastric disturbance* or exacerbation, though at times slight at first, or a previous so-called dyspepsia. Moynihan's and Rodman's views are noted below. Undoubtedly a history of former gastric ulcer, in some cases years before, can often be obtained. Kuttner[1] reports that 30 per cent. of his cancer patients had suffered for years from stomach trouble. Belching occurs, also more or less loss of sleep and loss of strength—the symptoms looking much like a mild gastritis. The tongue is usually thickly coated. The symptoms gradually become more marked. Rarely the attack begins more acutely. The feeling of discomfort gradually merges into pain. This is generally not of the severe spasmodic type of ulcer, but is continuous in character, there not being the intermissions of freedom as in ulcer. It may remit somewhat. The pain is at times increased by the food, but is often intense at a later period after eating than in ulcer. With the belching there is at first regurgitation of food, later vomiting, usually not after every meal, but once or twice a day. This is a prominent symptom when the growth causes a stenosis of the pylorus.

I have had a patient at the Red Cross Hospital with carcinoma of the greater curvature and body of the stomach, an inoperable case who has never vomited at all, the motor function being fairly good.

Later, hematemesis occurs, generally several times in succession, and the vomitus is of coffee-ground appearance and not large in quantity. The tumor usually becomes palpable at this time, though often earlier. The patient has, meanwhile, been steadily losing weight, and this loss becomes more and more marked and anemia and cachexia are prominent. He becomes more weak and prostrated and finally dies of inanition or of complications.

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[1] Therapie der Gegenwart, Berlin, Jan., 1911, p. 411, No. 1.

On the other hand, Rodman[1] reports that within a year he has seen 5 cases in which it was practically certain that carcinoma of the stomach was preceded by a long-standing ulcer, in one instance for twenty-seven years, in another, nearly twenty years. Moynihan[2] states that in his last 22 cases of carcinoma of the stomach operated upon, 16 patients gave a previous history of gastric ulcer. In 2 cases a history of chronic indigestion, extending for over twenty-five years, was given. Hence, in 72.1 per cent. there had been a gastric ulcer years before. The shortest interval between the attack of gastric ulcer and the onset of the symptoms leading to operation was three years, the longest interval twenty-six years, while 2 patients gave a history of less than three months. In the same article Moynihan reports 17 additional cases of carcinoma upon which he operated, out of which 8 gave a previous history of gastric ulcer. These records show *that a careful anamnesis will often elicit previous gastric disturbances.*

These general symptoms are modified by the position of the growth. A brief analysis of the symptoms is advisable.

*Anorexia,* or loss of appetite, occurs in about 85 per cent. of the cases, and it seems, as a rule, to be progressive. There is at times a special aversion to meat. Riegel believes that in the early stages, while the motor power remains undisturbed, the appetite remains good. This would seem as if toxemia were a factor. Boas reports fair or increased appetite in some cases, believeing loss of appetite to be due to lack of care of the mouth and tongue. The toxemic theory of loss of appetite seems most logical.

*Pain.*—Pain is the most *constant symptom,* Osler reporting it in 130 out of 150 cases; Brinton finds it in 92 per cent. of his patients, and others report a higher percentage. It usually begins at an early date, generally in the epigastrium, but may be referred to the hypochondriac regions, the sternum, or sometimes extends to the shoulders or back. It may be lancinating or of a dull, gnawing, or burning character. There may be tenderness on pressure in the epigastrium. The pain does not occur in paroxysms and is not relieved by vomiting, as in ulcer. It is continuous and never entirely disappears, though it may remit. As a rule, it is less intense than with ulcer. It is not relieved at the end of gastric digestion. In some cases it may occur more markedly after eating, though it is not especially influenced thereby. In other cases there may be more of a painful dull feeling, and it is not circumscribed as in ulcer. Sometimes over the region of the tumor the pain is most intense, as is the tenderness. Head believes there are areas of skin tenderness between the nipple and umbilicus in front and the fifth to the twelfth dorsal vertebræ behind.

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[1] Jour. Amer. Med. Assoc., Jan. 18, 1908, pp. 165–169.
[2] Brit. Med. Jour., Feb. 17, 1906, p. 370.

Exacerbations of pain are caused by ulceration of the growths or by formation of adhesions.

Moreover, Erlanger[1] describes cases in which attacks of pain diagnosed in some cases as intercostal neuralgia were the first or the principal sign of gastric cancer. The pain radiated to the shoulder in one case. One must think of the possibility that neuralgic pains may be due to the cancerous involvement of the regions innervated by the intercostal, lumbar, or sacral nerves.

*Vomiting.*—It may come on early, but more usually later. Osler reports it in 128 cases out of 150; and Brinton in 87 per cent. At first it occurs at rather long intervals, but later may be present several times a day. It is more frequent *when the pylorus is involved*, and may come on some hours after the ingestion of food, or at times on rising, when there may be mucus or undigested food in the vomitus. It may be offensive in odor or contain changed blood, micro-organisms (Boas-Oppler bacilli), and isolated yeast-cells; but rarely sarcinæ, which are *most common in benign stenosis.* Vomiting can occur, even if the orifices are not involved; if the cardia is affected, then regurgitation is characteristic.

Extensive involvement of the anterior or posterior wall or fundus may be present without vomiting.

*Hemorrhage* occurred in 36 of Osler's cases out of 150; while Brinton places it at 42 per cent. Riegel believes these percentages too low, as hemorrhage may be occult, or often not examined for in the vomitus or stool. The blood is occasionally ejected in sufficient quantity to be visible. It is more frequently mixed with gastric juice, food, and mucus, and presents a blackish, brownish, or coffee-ground appearance; is rarely bright red, Osler finding it in 3 cases. The quantity is much less than with ulcer, though frequent small hemorrhages may occur. Blood in the stool (melena) may accompany it, though this is rarer than in ulcer. A fatal hemorrhage seldom occurs.

*Loss of Weight and Cachexia.*—Progressive emaciation, especially if the disease is running a rapid course or has existed for some time, is a consistent feature. In the early stages we may occasionally see patients who appear fairly healthy or have little loss of weight, or at times temporary improvement may occur under treatment. Unfortunately, this is evanescent, and progressive emaciation takes place. In the later stages this is marked, as are the sallow skin and peculiar ashy and cachectic appearance, with loss of strength proportionate to the loss of weight.

*The Blood.*—Anemia is always present, and usually marked and progressive; when pyloric stenosis occurs with dilatation and insufficient water absorption, on account of the concentration of the blood the number of red cells may not be greatly reduced.

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[1] Archiv für Verdauungs-Krankheiten, Berlin, 82.

The average count in 59 of Osler's cases was 3712, 186 per cmm. Average of the hemoglobin was 44.9 per cent.

Schneyer[1] has shown that normal digestive leukocytosis is absent in gastric cancer, and that the number of leukocytes during digestion and fasting is the same. Osler claims that only 54 per cent. gave positive reaction.

Leukocytosis is present in gastric carcinoma, usually of mild degree, and rarely above 12,000 to 15,000. Eosinophilia is usually present and suggestive. We find some cases without apparent tumor in which the blood count is so low as to be suggestive of pernicious anemia, but the absence of megaloblasts and the presence of leukocytosis speak for cancer.

*Tumor.*—In connection with the symptoms, the presence of a tumor in the gastric region is a reliable diagnostic point. In the early stage it often cannot be determined. If large and superficial, it is readily detected. In Osler's 150 cases, tumor was detectable in 115. The methods of determination are as follows:

1. *Inspection.*—Position should be dorsal and the knees flexed to relax the abdomen. In some cases a protrusion can be seen in the gastric region below the ensiform or at the margin of the ribs. If there is dilatation, the lower curvature of the stomach may appear as an arched line below the umbilicus, moving up and down during respiration. Peristaltic movements are present with stenosis. With gastroptosis, the lesser curvature may be seen at times and the tumor situated thereon moving during respiration. If the tumor is at the pylorus, it may draw the stomach downward and the protrusion may be seen low down in the abdomen or even at the pelvic brim. Pulsation from the aorta may be transmitted to the tumor. Intrinsic movements in the hypertrophied muscularis may cause the tumor to appear and disappear. A subcutaneous umbilical nodule can at times be observed.

Inspection with the patient standing, as suggested by Knapp, should be carried out in any case, as the tumor can at times be thus more readily appreciated. Frequently, simple inspection gives no information.

2. *Percussion.*—There is dulness on percussion, or a dull tympanitic note over the tumor, which can be differentiated from the surrounding tympanites. Auscultatory percussion for its determination is described and illustrated in Chapter V, Figs. 47–49.

3. *Palpation.*—This is usually quite reliable. It determines the position and size of the growth, whether hard and nodular or smooth, its respiratory motility, and whether it is painful.

It is difficult to recognize a tumor on the posterior stomach wall unless it is thin and the stomach empty. On the lesser curvature, with the stomach in the normal position, it can only be felt on forced inspiration.

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[1] Berliner klin. Wochenschr., 1894, No. 41.

4. *Respiratory Motility.*—Tumors of the curvatures show greater respiratory motility than those of the pylorus. When the latter is adherent to the liver, it follows the excursion of the diaphragm.

Tumors are *smaller to the palpating finger* than they are found to be on operation.

5. *Inflation.*—This is of value in aiding inspection and also in determining whether the growth is connected with the stomach, if it be adherent, and in some cases the position of the tumor in the organ. Air can be employed for the purpose, or, more easily, carbonic acid gas, by the method already described.

If the tumor lies in close contact with the liver and moves away from it during inflation, the diagnosis of tumor of the stomach is evident, and the liver and gall-bladder are excluded. If this does not occur, there may be adhesions or involvement of both organs. If the tumor changes its position during inflation, there are probably no marked adhesions with the neighboring organs, an important fact in reference to operative procedure.

A tumor of the pylorus generally moves to the right and downward on inflation; and if held in this position by the hand will not ascend during expiration—expiratory fixation (Minkowski). If adherent to the liver, it will move upward.

Tumors of the posterior wall and lesser curvature that are palpable before inflation are frequently no longer so thereafter. With gastroptosis, however, the tumor would be palpable, but lie higher up.

A tumor of the greater curvature descends when the stomach is inflated and occupies the lowest border of the area of inflation; it is freely movable on respiration.

The position of the tumor should be marked on the *abdominal wall before inflation* for a basis of comparison. Inflation of the colon sometimes aids in the location of the growth.

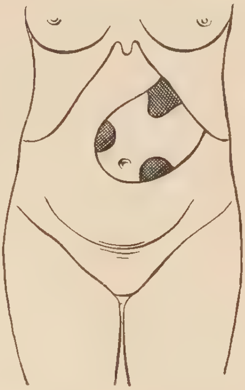

Fig. 158.—Composite from three patients showing dark areas in transilluminated stomach, produced by carcinomata of the curvatures and pylorus.

6. *Transillumination of the Stomach.*—This method is of value for the early recognition of tumors, but only if they lie on the anterior surface, on the curvatures, or at the pylorus. With the circumscribing gastrodiaphane the lesser curvature can be explored. The method with fluorescent media is preferable. The tumor being opaque, appears as a dark spot projecting into, or within, the transilluminated area; on top, when the lesser curvature is involved; below, if the greater curvature; to the right, if the pylorus (Fig. 158).

*Temperature.*—This is not a regular symptom, but often appears in the later stages. It occurred in 74 of Osler's 150 cases. It is of an intermittent type and rarely runs over 101° F. Chills have occasionally been associated. Fever is probably due to some inflammatory process, or to toxic absorption from the growth.

*Constipation* occurs in the majority of cases and is obstinate and marked; occasionally there is diarrhea, due to food deomposition or to scybalæ, causing irritation of the intestinal canal, or as a terminal symptom due to sloughing of the cancer.

*Coma* similar to diabetic coma may occur, and is believed to be due to acid intoxication.

*Thrombosis* of the femoral vein is an occasional symptom. Osler reports general thrombosis of the superficial veins in one case.

*Edema.*—Swelling of the ankles frequently occurs toward the close, ascites and general anasarca sometimes are present, and the latter may appear early.

*Metastases* have already been described. A small nodule appearing at or near the umbilicus, though rather rare, may aid in the diagnosis.

*Multiple neuritis* is an occasional complication.

*Perforation* is rare.

*Tetany* is a rare complication, but has occurred with malignant stenosis of the pylorus with ectasia.

*Urine.*—Excessive nitrogen excretion has been found in some cases, but it is not constant. Salkowski[1] and Kojo have worked out a method for determination of the nitrogen precipitated *by salts of the heavy metals* in relation to the total nitrogen output. The average in the urine of ten healthy persons was 1.22 per cent., while in ten cancer patients it averaged 3.03 per cent., the maximum being 4.62 per cent. The minimum, 2.15 per cent., was never reached in healthy urine. These facts are suggestive, but further investigation must be made to see if these findings are pathognomonic of malignant disease. Diminution of the chlorids is quite frequent, while indicanuria is common. Glycosuria, acetonuria, and peptonuria have been described.

Peptonuria indicates absorption from an ulcerated area. Nephritis is often present, but would be expected in advanced age.

**Special Symptoms Produced by the Location of the Growth.**—(*a*) *Cancer of the Cardia.*—One of the first and an important symptom is *dysphagia.* The patient finds that there is some impediment to the entrance of solid food into the stomach and assists it by drinking water. The condition gradually grows worse. Later, it is impossible to take solid food at all, as it sticks in the esophagus, causing much discomfort, and is finally regurgitated, often with considerable straining or retching. Finally, most of the fluid even is returned. There are often severe pains behind the end of the sternum and burning sensations. Mucus is sometimes ejected with the food, and occasionally blood. The cervical lymph-glands are often enlarged. Bronchitis or bronchopneumonia are frequent terminal events.

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[1] Berliner klin. Wochenschr., Dec. 12, xlvii, No. 50.

*Physical Examination.*—The stomach is of normal size or, in some cases, contracted; the swallowing sound is occasionally absent or heard in fifteen to twenty seconds instead of in the normal period of seven, though this is not invariably true.

Examination of the esophagus, *preferably with soft stomach-tubes of different sizes*, should be made. The position of the stricture can be noted by marking on the tube at the teeth when resistance to its passage is first encountered, and measuring the distance from the mark to the tip of the tube. *Smaller tubes* are then employed, until one of sufficiently small caliber is secured to enter the stomach.

In this way the degree of stenosis can be determined. Much force should never be used. In some cases it may be necessary to employ a stiffer tube of silkworm or bougies of varying sizes, of which Einhorn's divisible instrument is convenient (Fig. 159).

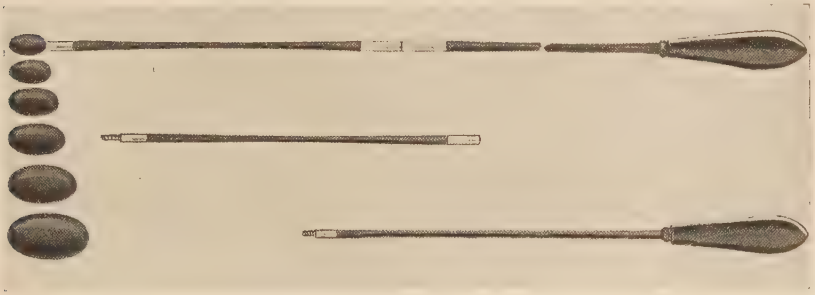

Fig. 159.—Einhorn's divisible esophageal bougie.

The soft stomach-tube, with openings at the tip and side, is of value for safety; by pinching the tube before withdrawal, blood, either fresh or decomposed, of foul odor or mixed with mucus, may be found within it, which, in conjunction with the other symptoms, is suggestive of malignancy. Occasionally a small tumor fragment may be removed, which should be submitted to microscopic examination. With malignant stricture of the esophagus, dilatation above the point of stenosis and *retention of food are associated.* It also occurs in benign stricture from syphilis or from traumatism from burns by acids or alkalis, or with congenital stenosis of the esophagus, a rare condition.

After the ingestion of suspended bismuth subcarbonate, examination with the *x*-rays and the fluoroscope will aid in determining the site of the stricture. This is of special value when it is not desirable to use the bougie. A röntgenograph should be taken. The complete methods of examination are described under Examination of the Esophagus and Esophageal Diseases.

Aspiration of the esophagus above the seat of stricture after a small test-meal, and then the passage of a smaller tube and aspiration of the stomach-contents, and a differential examination of the contents are not necessary for diagnosis, though they have been recommended.

The food removed from the esophagus would present the appearance as when swallowed, and there would be no hydrochloric acid; while in the stomach the particles would be finer, the reaction acid, and free hydrochloric acid might or might not be present. This is also true of the ferments. It has been demonstrated by Moore and Friedenwald[1] that, in cancer of other organs than the stomach, there may be diminution or absence of free hydrochloric acid in the gastric contents, and this occurs at times in cancer of the cardia. The presence of *stricture* and *dysphagia*, the age of the patient, and the general symptoms are diagnostic.

With diverticula of the esophagus, which usually occur at the junction of the pharynx and gullet, there is generally a swelling in the neck, which can be diminished by pressure (the contents expressed), and symptoms of cancer are absent. (See also Examination of Esophagus.)

Spasm of the esophagus usually occurs in nervous patients, and the tube when passed is temporarily arrested. Larger tubes often pass more readily than the small ones. There are no symptoms of malignancy. With persistent cardiospasm, fusiform dilatation of the esophagus may result. This has been referred to and the röntgenograph depicted.

(*b*) *Cancer of the Pylorus.*—The chief subjective symptoms are pain, a full feeling in the stomach and other dyspeptic symptoms, and frequent attacks of vomiting. Just before emesis there are often severe exacerbations of pain, due to the contractions of the stomach and the effort to expel the contents through the pylorus.

The vomitus is generally large in amount (1 to 2 liters) and may consist of food taken the day before. Motor insufficiency is marked, the contents on aspiration being found to consist of more or less decomposed food, when the fasting stomach is examined. The particles of food are often quite large and obstruct the openings of the tube, and are difficult to remove by lavage.

*Physical Examination.*—Peristaltic unrest is a frequent symptom. Dilatation of the stomach is present, as determined by the methods of examination described. Gastroptosis may be present. Frequently a tumor can be detected lying in the epigastrium to the right of the median line or, if ptosis is present, at a lower level.

(*c*) *Cancer of the Body of the Stomach.*—Pain, anorexia, and other symptoms are manifested. Vomiting occurs in some, but in many *cases is absent.* The vomitus contains food and occasionally coffee-grounds, the food is more finely divided.

The tumor frequently lies to the left of the median line; but if on the upper curvature or posterior surface of the stomach, is not always detectable.

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[1] N. Y. Med. Jour., Aug. 24, 1907.

There is motor insufficiency of a slight or moderate degree due to infiltration of the muscular tissue by the growth, and occasionally, if the tumor be large and on the greater curvature, a slight dilatation.

**Laboratory Diagnosis.**—In conjunction with clinical symptoms, analysis of the gastric contents and microscopic examination aid in establishing the diagnosis of cancer of the stomach.

*Gastric Contents.*—Golding Bird, in 1842, first refers to the diminution or absence of hydrochloric acid in gastric cancer, but Von der Velden, in 1879, first studied the question in a scientific manner.

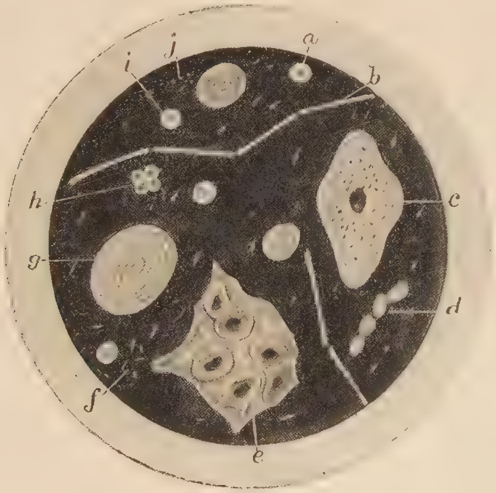

Fig. 160.—Gastric contents in carcinoma. Dark ground illumination × 460; *a*, Leukocyte; *b*, Boas-Oppler bacilli; *c*, squamous epithelium; *d*, yeast; *e*, mucous membrane fragment with carcinomatous cells as rarely found in wash-water; *f*, bacilli; *g*, wheat starch grain; *h*, sarcinæ (very rare); *i*, fat-droplet; *j*, cocci.

For *accuracy* the vomitus should be examined; analysis of the gastric contents and microscopic examination, after the test-breakfast, should be made and the stomach washed out. If small fragments of mucosa are found, these should be examined.

*Vomitus.*—Macroscopically, undigested meat-fibers and coarse food particles are found; the quantity is variable, depending upon the motor functions. Coffee-ground material is present in many cases and often a foul odor in advanced cases.

Microscopically, undigested muscle-fibers, remnants of vegetable material, starch granules, fat-droplets, numerous fungi; *sarcinæ are rare;* yeast-cells are found in stagnating contents, though usually as isolated specimens; and blood and Boas-Oppler bacilli (Fig. 160).

Boas and Strauss report pus[1] in some cases. Palier[2] holds that staphylococci are present in the gastric contents of cancer.

Occult blood should be tested for by Weber's or the benzidin or aloin test, if none appear microscopically.

*Examination of Test-breakfast.*—Ewald's or Boas' test-breakfast should be given, and one hour later the contents aspirated and examined. Repeated analyses should be made.

1. *Hydrochloric Acid.*—Out of 94 of Osler's cases of gastric cancer, in 84 free hydrochloric acid was absent; and in 40 cases, Boas found it absent in 35; while Graham and Guthrie found in 150 cases of cancer of the stomach in the Mayos clinic that free hydrochloric acid was absent in only 80 cases. In the first stages of gastric carcinoma, however, free HCl is diminished, not absent. A progressive diminution in the percentage of free HCl during the course of a month or more, as determined by frequent examinations, when taken in consideration with the other symptoms, is corroborative of cancer. Einhorn reports 6 cases of gastric cancer in which free hydrochloric acid was present in normal or excessive quantity.

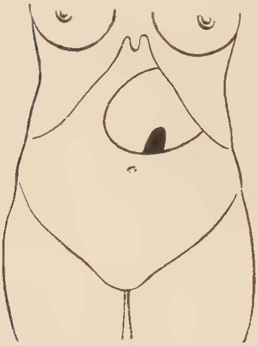

Fig. 161.—Irregular type of carcinoma under trypsin treatment at Red Cross Hospital. Carcinoma of stomach, anterior wall involving greater curvature; slight reduction of motor function. Tumor evident by palpation, percussion, and gastrodiaphany. History of ulcer with carcinoma engrafted. Hyperchlorhydria present.

In gastric cancer, engrafted on gastric ulcer, free HCl (or hyperchlorhydria) has been noted in the earlier stages, often with progressive diminution accompanying the increased development of the cancer. I have seen hyperchlorhydria persist in quite an advanced stage, notably in a case at the Red Cross Hospital, which was deemed inoperable. The patient, aged sixty-eight, suffered with the symptoms of gastric ulcer for a year, falling off in weight from 240 to 200 pounds. There was then a slight gain. Later, he lost steadily, and when admitted to the hospital weighed 149 pounds, was extremely weak and cachectic, with a tumor on the anterior surface of the stomach, involving the greater curvature, and with the clinical symptoms of carcinoma, except the gastric findings, which were hyperacid, with a total acidity of 90+, free hydrochloric acid 70+. The position of the growth is shown in Fig. 161. There was only slight disturbance of motor function and no vomiting. Anemia, leukocytosis, and eosinophilia were present.

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[1] Pus may be found in benign ulceration, as in Connors' case, as well as in malignant ulcer.

[2] N. Y. Med. Record, Nov. 19, 1904.

The patient gained 20 pounds in weight under treatment and proper diet, and was able to go out daily. From his *ultimate experience with trypsin*, the writer was obliged to conclude that the diet and treatment of the hyperacidity were the main factors. His strength markedly improved, as well as his appearance. The growth did not disappear. The patient returned to work and at the end of several months contracted pneumonia and died.

We know that free hydrochloric acid is markedly *diminished*, or even *absent*, in severe catarrhal gastritis, and is absent in achylia gastrica. Moreover, hypochlorhydria (diminished acidity) or even absence of free hydrochloric acid *per se* are not always of diagnostic significance. Kelling[1] gives the findings in 4937 stomach cases, and in these, excluding ulcer, every seventh man and fifth woman had no free hydrochloric acid. This condition was frequently associated with disease of other organs. No free HCl was found in 30 per cent. of 254 cases of gall-stones, and in from 30 to 40 per cent. of diabetes, gout, renal calculus, and tuberculosis. Diagnosis was only possible after the test-breakfast. The *absence* of HCl is not *pathognomonic of cancer*, but taken in conjunction with the clinical symptoms is confirmatory.

2. *Lactic Acid.*—It has been known for some years that organic acids were increased in cancer of the stomach and lactic acid was present; but to Boas must be given the credit of attaching diagnostic significance to it, and who first described exact quantitative and qualitative methods.

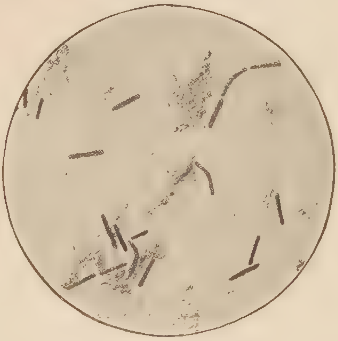

Fig. 162.—Boas-Oppler bacillus from near top of fluid from washing in case of gastric cancer. Observation at Pennsylvania Hospital (Boston).

He washes the stomach and gives a plate of barley soup, which contains no lactic acid, and an hour later aspirates the contents. Lactic acid should be examined for by Uffelmann's test or by Boas' method. Ewald's test-breakfast will often suffice for practical purposes.

In most cases lactic acid is present in considerable quantity, though occasionally it is absent when free hydrochloric acid is in evidence. In non-malignant stenosis with dilatation it has been found, so it cannot be said to be *absolutely pathognomonic*. The absence of free *hydrochloric acid* and the *presence of lactic acid* are confirmatory, in conjunction with other symptoms.

Boas-Oppler bacilli are found. These are rods of considerable length, frequently joined at their ends, and form long angulated threads, stainable by methylene-blue or other anilin dyes (Fig. 162). They must be distinguished from the Leptothrix buccalis (found in the mouth). A drop or two of Gram's solution should be added to the specimen. The Boas-Oppler stains brown with the iodin, the leptothrix, blue.

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[1] Archiv. für Verdauungskrankheiten, Oct., 1909.

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Gram's solution consists of iodin, 1 part; potassium iodid, 2 parts; water, 300 parts.

The Boas-Oppler bacillus is found in about 80 per cent. of cases of carcinoma of the stomach; also rarely in non-malignant stenosis with dilatation of the organ.

With absence of HCl, an alkaline medium, and absence of stagnation, Paul Cohnheim also holds that the Trichomonas hominis, Megastoma entericum, with associated amebæ, pus, and blood, are found in carcinoma of the stomach, not affecting its motility (see p. 140).

*Pus.*—The detection of pus in the gastric contents by microscopic examination is of diagnostic value. The presence of blood, or occult blood, is also important before the tumor is palpable.

**Diagnosis.**—*Glycyltryptophan Test for Carcinoma of the Stomach.*—This new test, devised by Neubauer[1] and Fisher,[2] in December, 1909, depends on the fact that the digestion of proteins proceeds further in a carcinomatous stomach than in a normal one. This is due to the presence of an enzyme from the carcinoma, which exhibits strong proteolytic powers, and which, like trypsin, is capable of converting proteins as well as simple peptids into amino-acids. Pepsin can only digest proteins to albumoses and peptones. These investigators have utilized these facts, and use an amino-acid, tryptophan, which in peptid combination, as glycyltryptophan, cannot be digested by pepsin and does not give a color reaction with bromin, but when the tryptophan is split by tryptic ferments (carcinoma) a reddish-violet color or reddish-pink results, when bromin vapor is added.

**Method.**—An Ewald's test-breakfast is administered and extracted in thirty to forty-five minutes, preferably the latter. It is better to employ water as the fluid part of the test, instead of tea, which last, on account of its tannic acid, inhibits blood reactions.

A small portion of the aspirated contents is then tested for occult blood and bile, by the usual methods. If the results are negative, the contents are filtered and a small portion is tested for tryptophan with bromin vapor. If this also is negative, the following procedures should be carried out: Add 10 c.c. of the filtrate to 2 c.c. of glycyltryptophan solution. The mixture is covered with a layer of toluol and placed in an incubator for twenty-four hours at 37° C. At the end of this time about 3 or 4 c.c. are removed from the bottle with a pipet and placed in a test-tube. A few drops of a 3 per cent. solution of acetic acid are added and bromin vapor is then blown gently down the side of the test-tube, in small amount at a time, so as not to color the filtrate too quickly. If *tryptophan is present*, a color develops which varies from a *light pink to a deep red violet*. In employing the bromin vapor it is preferable to add small successive quantities, thus giving the pink color an opportunity to develop, than to use a large amount at once, as an excess of bromin vapor bleaches the color produced by tryptophan and destroys the possibility of a positive reaction. In a negative test there is no color up to the point when the solution becomes saturated with bromin and turns yellow. To detect a faint pink, a control should be made using the same background. A saturated aqueous solution of bromin can be added, drop by drop, from a fine pipet to the solution instead of employing the bromin vapor. A maximum of 5 to 7 drops may be required.

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[1] Bull. Johns Hopkins Hosp., 1911, xxii, 150.

[2] Deutsch. Arch. für Klin. Med., xcvii, 449, 1909.

Glycyltryptophan is a clear watery solution. It is dispensed by Kalle & Co. ready for use in little bottles. Each bottle contains a few minims of glycyltryptophan solution, with a little toluol over it as a preservative. The bottle has a mark on it up to which the filtered gastric contents is to be added. The total capacity is about 15 c.c. The filtrate is allowed to flow from a pipet inserted below the toluol, and there mix with the glycyltryptophan. A second test has been suggested by Neubauer. A fresh half-saturated solution of calcium chlorid is diluted to one-fifth its strength, and added drop by drop instead of bromin vapor. A rose color shows positive reaction.

There are certain factors in the test which may lead to error, according to Neubauer and Fisher. They are as follows:

1. The presence of tryptophan in the stomach-contents. This is not frequent. Regurgitation of intestinal contents or marked stenosis may favor its development. If present, the specimen should be rejected and another specimen tested.

2. The presence of blood in the stomach-contents may split up the peptid glycyltryptophan, as blood contains a proteolytic ferment. If occult blood is present, as shown by the guaiac or benzidin test, the specimen should be discarded. Lyle[1] has demonstrated that dilutions of blood weaker than 1 : 1000 will not interfere with the test, and it is doubtful if 1 : 500 will do it. Specimens containing a higher per cent. of blood cannot be used.

3. The presence of trypsin in the stomach-contents. Neubauer and Fisher exclude this possibility by examining the stomach-contents in gross for bile; Lyle, however, shows that this method is not reliable, as trypsin may be present when tests for bile are negative. To prevent the possibility of regurgitation of intestinal contents, Lyle advises a strong laxative the night before and that the test-meal after *free purgation be given the following afternoon.*

4. The presence of peptid-splitting bacteria. Some species of bacteria may split off tryptophan. Filtering may remove the organisms, but the addition of toluol prevents this occurrence if the tests are performed within twenty-four hours.

5. The presence of free hydrochloric acid interferes with the test. Abderhalden[2] has shown that tryptic ferments are quickly killed by

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[1] N. Y. Med. Jour., June 4, 1910.

[2] Zeit. f. Physiol. Chemie, lvii, p. 317.

normal acidity, and Neubauer and Fisher state that 0.36 per cent. of hydrochloric acid will render the test negative. They advise that if more than this percentage of acid is present, the filtrate should not be used, but another test-meal should be extracted with which an alkali was given at the time of ingestion.

Lyle and Kober[1] advise care *in drawing negative* conclusions in cases where the acidity is over 12+ to 15+, as the carcinoma ferment may have been destroyed by it. They also advise that the HCl acidity of the filtrate should be immediately neutralized by magnesia usta after withdrawal, before adding to the glycyltryptophan.

J. W. Weinstein[2] has experimented with this method. He holds that the use of glycyltryptophan is superfluous, and that it is only necessary to test for tryptophan in the gastric filtrate without using glycyltryptophan at all. The presence of tryptophan he claims is sufficient. He prefers[3] a regular dinner given at noon, consisting of bread and butter, meat prepared plainly without seasoning, and very sweet weak tea without milk. Pepper and lemon-juice interfere with the reaction. Sugar is an excellent stimulant to the secretion of the cancer enzyme and beef is better than chicken. Three or four hours later the gastric contents are aspirated and *tryptophan tested for;* if present, he deems reaction is positive. If negative, some of the filtrate is placed in the thermostat and tested for tryptophan, twenty-four or forty-eight hours later. When a negative tryptophan test is obtained in a case of pyloric stenosis showing free HCl, lavage may be instituted and Ewald's test-breakfast given, in hope that contents may be free of free HCl and that the reaction will be secured. Acetic acid and bromin vapor are employed.

Weinstein[4] holds that the only practical sources of error are: (1) usually the presence of free hydrochloric acid equal to 0.36 per cent., though he has noted exceptions; (2) the regurgitation of duodenal contents, so that the contained trypsin may produce tryptophan. He believes that bile and pancreatic secretion would appear together in the stomach, as do Neubauer and Fisher, and that occult bile could hardly be accompanied by sufficient trypsin to produce a detectable amount of trypsin. Sufficient duodenal secretion to vitiate the test he believes rare. He further disagrees with Fisher and Neubauer as to occult blood and bacteria interfering with the test.

Sufficient blood or coffee-grounds (blood) to color the contents, coffee, tomatoes, strawberries, strong tea, etc., sufficiently redden the contents so as to vitiate the test.

**Precautions.**—The reaction may be present in one specimen of gastric contents and absent in another from the same patient. At least three tests should be made, and observers agree that if these are all negative, the case is likely non-cancerous; *with a very low content or absence of free HCl* and *repeated positive tryptophan reaction*, a conclusion as to positive cancer seems reliable.

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[1] N. Y. Med. Jour., June 4, 1910.

[2] Jour. Amer. Med. Assoc., Sept. 24, 1910.

[3] A test-breakfast consisting of a glass of sweetened water, with some white bread or toast or biscuits can be substituted. A glass of milk can be added. Aspirate in one hour.

[4] Jour. Amer. Med. Assoc., Oct. 8, 1911.

Moreover, in cases reported by Lyle, Weinstein, and others, absence of free HCl and negative reaction for tryptophan has been proved by operation to demonstrate that no cancer was present in the stomach, but it occurred elsewhere, or some other condition was present, such as cholecystitis, etc. The *negative phase* has thus proved of value in excluding carcinoma of the stomach. Friedman[1] has tested this reaction and, though finding the reaction positive in many cases, does not look upon it as pathognomonic of cancer.

In the transition stage, the development of carcinoma from gastric ulcer—with hyperchlorhydria or even a moderate amount of HCl being present—this reaction would not be found, and hence for early diagnosis in these cases one must depend on the presence of occult blood, pus (microscopic), progressive anemia, loss of weight, and progressive weakness.

In the early stage of carcinoma, when no history of previous gastric ulcer can be secured and when there are *merely progressive anemia, loss of weight, weakness, loss of appetite*, and *gastric symptoms* with *considerable free HCl still present*, the glycyltryptophan reaction cannot be obtained. In a patient of *middle age such symptoms are suggestive of malignancy*. It is in this class as well as in the suspected transition cases, when frequently no tumor is palpable, that the author believes *exploratory laparotomy for the purpose of diagnosis is indicated.* The writer believes that *not until the medical profession can be educated to this view point will we secure the early operation for and cure of carcinoma of the stomach.*

*Antitryptic Reaction of the Blood in Cancer.*—Brieger and Trebing[2] have announced that they have determined a new characteristic of the blood in cancer—namely, a marked increase in the power of the blood-serum to inhibit the proteolytic activity of solutions of trypsin. Fuld and Gross[3] employ a solution of casein as a medium of digestion, thus differing in technic, while Feldstein and Weil[4] employ the viscosity method. All observers agree that the great *majority of cases of cancer* give evidence of *increased antitryptic value in the serum*. This condition, however, has been frequently found in the acute infections, such as pneumonia, typhoid fever, sepsis, and polyarticular rheumatism; in chronic infections, notably, tuberculosis; in diabetes and severe anemias; and in Graves' disease almost constantly. The reaction occurs with the inauguration of artificial feeding in infants and with the onset of labor and persists through the puerperium.[5] The increase in the antitryptic index cannot, therefore, be called a specific symptom, but, on the other hand, the *absence of the antitryptic* reaction in the blood may be taken *generally as arguing against the existence of cancer.*[1] A positive reaction, excluding the conditions noted, argues the probability of cancer.[2]

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[1] Archiv. of Diag., New York, April, 1911.

[2] Berl. klin. Woch., 1908, p. 1041.

[3] Deutsch. Arch. f. klin. Med., 1908, p. 319.

[4] Proc. Soc. Exp. Biol. and Med., Feb., 1910.

[5] Becker, Münch. med. Woch., 1909, p. 1363.

*Hemolysis as a Diagnostic Method in Cancer.*—By recent researches they have attempted to demonstrate that there exists in the blood-serum of patients affected with cancer a certain substance or substances which are capable of causing destruction of the red blood-cells of patients not cancerous. Kelling first commented on this occurrence, and Crile apparently sets store by the method. Weil has experimented with it, and Smithies[3] has recently reported on the subject. It will be noted that the reaction occurs with tuberculosis, anemia, syphilis, and occasionally with normal subjects. The author does not believe it furnishes accurate information. Weil believes the method not diagnostic, but a positive reaction may be cautiously considered as an auxiliary to the diagnosis.[4]

*A Skin Reaction in Cancer.*—Based on the same hemolytic action of the serum of cancerous patients, Elsberg[5] and Neuhof conceived the idea of bringing the red cells into immediate contact with the serum of the cancerous patient *in vivo.* A suspension of crushed erythrocytes in saline solution was injected under the skin of both normal and cancerous patients. About five hours after the injection a reaction appeared, consisting of an oval, irregular reddish area from 2 to 5 cm. long, and from 1 to 3 cm. wide, at times surrounded by a white areola. This increased in intensity for two to three hours, and persisted for six to twelve hours. In 432 individuals, 684 injections were given. In 89.9 per cent. of the cases in which reaction occurred, there was a positive diagnosis of cancer, and in 94.3 per cent. of cases with no reaction it was possible to exclude malignant disease. It is a question whether this reaction would occur with other conditions as with the experiments described by Smithies.

Gräfe and Röhmer[6] claim there are *hemolytic substances in the stomachs* of cancer patients. This never occurs in the healthy subject except in the presence of trypsin, when the test is of no value. A negative test excludes malignancy, a positive test shows an ulcerating surface, cancer, or ulcer.

In a patient of forty-five to sixty or more years of age, suffering from recent gastric disturbances of six months' or a year's duration, or less, with a *previous history of good health or of former gastric ulcer*, there being continuous pain, frequently *epigastric rigidity*,[7] *rapid loss of weight and strength*, anorexia with or without vomiting, motor insufficiency moderate, or great with marked ectasia, with diminution or absence of free hydrochloric acid, lactic acid present or absent; Boas-Oppler bacilli present or absent; whether or not there be tumor detectable, or whether or not coffee-grounds are present in the vomitus, such symptoms should be considered diagnostic of carcinoma of the stomach. As already noted, the presence of pus and occult blood are important.

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[1] Weil, The Antitryptic Activity of Human Blood-serum Amer. Jour. Med. Sci., May, 1910.

[2] Jour. Amer. Med. Assoc., Oct. 29, 1910.

[3] Med. Rec., Nov. 17, 1909.

[4] Jour. Amer. Med. Assoc., Oct. 29, 1910.

[5] Med. Rec., Oct. 15, 1910.

[6] Deutsch. Arch. für klin. Med., xciv.; also Med. Rec., Aug. 19, 1911.

[7] Anders, N. Y. Med. Jour., Nov. 21, 1908.

The Mayos now frequently send a patient to the operating-room with a diagnosis of cancer on an old ulcer, based on the history of a chronic ulcer with *recent exacerbation, accompanied by loss of weight, weakness, and anemia.* They do not wait for cachexia, tumor, and the text-book test-meal.

Associated with the presence of pus and occult blood when found, and the progressive anemia and loss of weight, together with loss of appetite and gastric symptoms already noted, the Soloman test when present may occasionally be of service in the diagnosis of an early case.

The method consists in washing out the stomach in the evening, and then on the following morning the introduction of 400 c.c. of normal salt solution. This is aspirated out after a short stay in the stomach and tested for the presence of albumin and for the proportion of nitrogen.

Marked turbidity with Esbach's reagent (picric acid) or the presence of more than 30 milligrams of nitrogen indicates the presence of carcinoma.

The glycyltryptophan test, as described, can also be applied when the HCl content is low, or the tryptophan test.

*Urinary Methylene-blue Reaction with Cancer.*—Fuhs[1] makes a preliminary report in which he claims that methylene-blue is decolorized by the urine of patients suffering from cancer. Three to five drops of Löffler's methylene-blue is added to a test-tube of the patient's urine, or sufficient to give it a decided blue color. The urine is shaken and allowed to stand for twelve to twenty-four hours at the room-temperature. A control fresh specimen of normal urine is similarly prepared. The blue color of the urine of the patient disappears at the end of twenty-four hours, except the color of the upper layer in contact with the air. The normal urine retains its blue color. The urines of pregnancy, rheumatism, nephritis, meningitis, etc., particularly of pregnancy, decolorize, but in most cases the test is negative in subsequent examinations as the condition of the disease improves, while with malignant disease the change is constant. Verbrycke,[2] after a series of tests, holds the method of *no value.*

*Röntgen Rays.*—Anders[3] and Pfahler[4] hold that the *x*-rays aid in the early diagnosis of gastric carcinoma. The new growth makes itself evident by a change in the contour of the stomach wall; by disturbance of the peristaltic waves at certain points; at times by rigidity and contraction of the stomach wall; by adhesions which prevent the free motility of the stomach when the abdominal walls are contracted, or when the position of the patient is changed; and finally, in some cases, by obstruction to the passage of food. The method requires great care even by an expert, and may be an aid in connection with the clinical symptoms and physical and clinical examination (p. 107).

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[1] Jour. Amer. Med. Assoc., June 24, 1911.

[2] Med. Rec., Oct. 28, 1911.

[3] N. Y. Med. Jour., Nov. 21, 1908.

[4] Ibid., Aug. 21, 1909.

*Boas-Oppler Bacilli in the Stool.*—Neusser and R. Schmidt hold that Boas-Oppler bacilli are more easily found in the stool than in the gastric contents and aid early diagnosis of cancer. Gram-negative stools exclude cancer of the stomach; Gram-positive stools, uniform-sized Gram-positive bacilli (Boas-Oppler bacilli), show cancer.[1] I believe the Bacillus aërogenes capsulatus are found, and their deductions probably erroneous. Anaphylaxis[2] from injection of cancerous gastric juice into animals; the increase of amino-nitrogen in the gastric contents, and marked urine pepsin have been suggested as tests for cancer. They are uncertain.

**Differential Diagnosis.**—*Apparent Tumors of the Stomach.*—Prolapse of the left lobe of the liver, or a pulsating aorta, or thickening of part of the abdominal muscles (recti) are referred to by Einhorn as being mistaken for a tumor or possibly for a carcinoma of the stomach. In view of the fact that with these conditions gastroptosis is usually associated, the history is a long one, emaciation is of long duration, and the symptoms of cancer are absent, the mistake can hardly occur. They are also apt to be present in younger patients. Simple adhesions of the stomach give a history most frequently of gall-bladder disease, or gastric ulcer, or localized peritonitis. Adhesions are frequently present with cancer, but the symptoms and gastric findings are of carcinoma.

*Grave Anemia in Carcinoma Ventriculi, without Palpable Tumor.*—These cases must be *differentiated from pernicious anemia.* The type occurs with mild dyspeptic symptoms. The blood-count is rarely below 2,000,000 per cubic millimeter; there is absence of megaloblasts and leukocytosis is present, which speak for cancer. There is a lower color-index, as in secondary anemia.

In addition, the acidity of the gatsric contents is higher in cancer than with the achylia gastrica of pernicious anemia, and lactic acid is present in cancerous anemia. If, therefore, we have *gastric symptoms,* rapid loss *of weight,* and severe *anemic symptoms in an elderly patient,* the diagnosis of cancer is most probable. With secondary anemia or chlorosis, hyperchlorhydria is usually associated.

*Syphilis.*—This may present symptoms which may simulate carcinoma of the stomach, unless thorough examination be made. I have seen three types of this class.

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[1] P. K. Brown, Jour. Amer. Med. Assoc., Nov. 6, 1909, p. 1525.

[2] Med. Rec., Aug. 19, 1911.

1. *Sclerosis of the Stomach.*—A male patient, aged sixty, had lost 30 pounds in weight within a period of a year; was emaciated and weak, suffering from gastric symptoms and constipation. On examination, a small hard mass was found in the epigastrium at the left border of the ribs (Fig. 163).

It was demonstrated to be a hard and contracted stomach, giving the feel of a diffuse scirrhous carcinoma of the organ. The liver was diminished in size and hard. Free HCl was absent, the findings were of achylia gastrica. Syphilitic scars were in evidence and the patient acknowledged syphilis and alcoholism. The demonstration of Wassermann's reaction is diagnostic in doubtful cases.

2. *Cirrhosis of the Liver (Left Lobe—Syphilitic).*—Male, aged fifty-five, had lost 20 pounds in a few months and suffered from gastric symptoms. A hard mass could be felt in the epigastrium, extending down from the lower border of the left ribs, apparently a tumor of the lesser curvature of the stomach involving the anterior wall. Respiratory movements were present. Deep palpation elicited a free edge, and gastrodiaphany showed an opaque mass, but on moving the instrument a lateral view demonstrated the mass overlapping the stomach (Fig. 164).

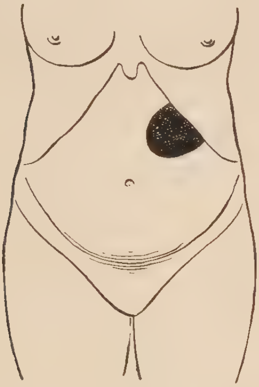

Fig. 163.—Syphilitic sclerosis of stomach. Physical examination of stomach suggests scirrhous carcinoma (diffuse) of stomach.

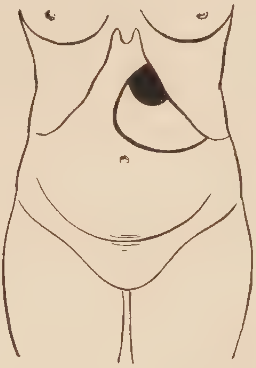

Fig. 164.—Syphilis. Enlargement of left lobe of liver overlapping stomach. Transillumination shows mass, which is readily palpable. Side view (gastrodiaphany) shows the mass not connected with the stomach. Free HCl trace; chronic gastritis.

Gastric findings were a trace of HCl and much mucus (chronic gastritis). Syphilitic scars and history were elicited. Improvement followed treatment directed to the stomach and syphilis.

3. *Syphilitic Stenosis of the Pylorus, Due to Gummatous Tumor, Simulating Malignancy.*—Male patient, aged thirty-eight. Weight, September, 1907, 196 pounds. At this time, anorexia, pain con-

tinuous in character, nausea, and occasional vomiting began. These symptoms gradually grew worse and the patient steadily lost weight. Early in November he entered a local hospital, where he was under treatment by lavage, etc., for eight weeks. His weight on leaving the institution was 137 pounds. He spent two months at a western sanitarium, where he grew steadily worse, having vomited small quantities of blood on several occasions. He was given morphin for pain and subsequently contracted the habit.

At the end of March, 1908, or about six and a half months after the first appearance of symptoms, he entered the Red Cross Hospital. His weight was 119 pounds, a loss of 77 pounds, epigastric pain nearly continuous, increased some after eating, tenderness in the epigastrium, peristaltic unrest of the stomach, dilatation to one finger below umbilicus, daily vomiting. There were marked emaciation and considerable anemia, but not the cachectic appearance of cancer. No tumor was detectable by palpation or transillumination, but a sense of resistance at the pylorus. Frequent gastric analysis invariably showed free HCl absent and abundant lactic acid. Morphin was shut off, and lavage, diet, and careful observations were carried out. A syphilitic history was secured.

On account of the age of the patient, the syphilitic history and the absence of true cachexia, in spite of the gastric findings, I believed the stenosis to be non-malignant (syphilitic) and advised operation. This was duly performed. There was a hard mass on the posterior wall of the pylorus, about the size of an English walnut, blocking the passage; no glandular involvement (Fig. 165). As the patient was in poor condition, rapid gastro-enterostomy was performed. He vomited once after operation. Mercurial inunction and iodids were given, and the patient left the hospital in good condition. He would unquestionably have died from inanition unless stomach drainage had been instituted, in spite of specific treatment.

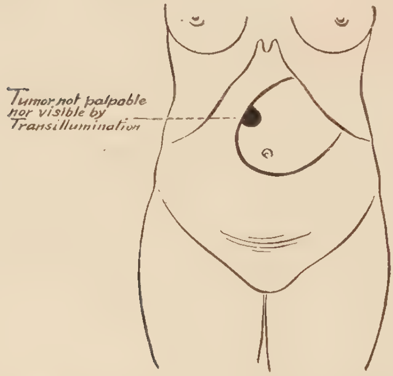

Fig. 165.—Syphilitic stenosis of pylorus due to gumma simulating malignancy. Transillumination shows dilatation, but no tumor. Growth found at operation lying on posterior wall of pylorus. Gastric findings of carcinoma, but no cachexia.

*Cancer Engrafted on an Ulcer.*—This condition, I believe, is a quite frequent occurrence. There is generally a gradual change in the symptoms and character of the gastric secretion, a gradual diminution of free HCl, though Riegel has brought to our attention that in this type the excessive production of hydrochloric acid *often* *persists for a long time*, and in *some rapidly fatal cases* may be present *until death.*

In these cases the diet and medication directed to the ulcer fail as soon as the malignant condition sets in. The pain increases and is continuous. There is a dislike or even loathing for food. There is rapid loss of weight and the typical cachexia appears—a change from the facies of ulcer. Hematemesis is more frequent, especially as occult hemorrhage, and not in the larger amounts, such as with ulcer.

This type should be considered in order to avoid error. I have already referred to a patient of this class (hyperchlorhydria), with a marked growth, cancer engrafted on an ulcer.

*Aneurysm of the Celiac Simulating Carcinoma of the Pylorus.*—The possibility of this error is interesting. A negro patient, aged forty-five, was seen by me at Roosevelt Hospital several years ago at the request of William H. Thomson.

The illustration (Fig. 166) shows the position of the stomach by inflation and that of the mass by palpation, it not being visible by transillumination. The tumor disappeared on inflation, diagnostic of a posterior position.

There were vomiting, gastric symptoms, such as pain, anorexia, etc., and a loss of weight of 40 pounds in three months. The patient was very weak. Dilatation to below the umbilicus. Gastric analysis; no free hydrochloric acid; lactic acid was present.

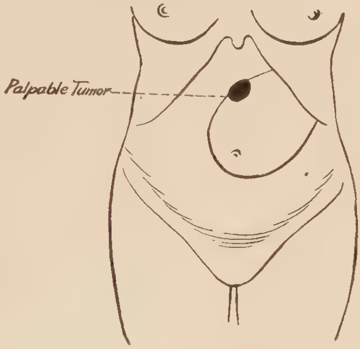

Fig. 166.—Aneurysm of celiac axis with symptoms simulating carcinoma of pylorus. No tumor visible by transillumination. Tumor disappears on inflation.

A palpable tumor, the size of a small egg, was present in the epigastrium, in which there was slight pulsation, but no bruit or thrill. From the history and results of examination it seemed to be a carcinoma on the posterior wall, involving the pylorus, with pulsation transmitted from the aorta.

The possibility of aneurysm was considered. Exploration by G. Brewer showed an aneurysm of the celiac axis pressing on the pylorus posteriorly. The resulting occlusion of the pylorus and circulatory disturbances from the aneurysm were responsible for the ectasia and changes in the gastric secretion. W. L. Rodman[1] reports a case of apparently a palpable cancer of the pylorus, with absence of free hydrochloric acid and the presence of lactic acid in the gastric contents. The diagnosis of carcinoma of the stomach had been made in several hospitals. Aneurysm of the abdominal aorta was found at operation.

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[1] Jour. Amer. Med. Assoc., Jan. 18, 1908, vol. i, pp. 165–169.

Syphilis and aneurysm must thus be considered in our differential diagnosis.

The following are the chief diagnostic points between cancer of the stomach and other conditions:

*Cancer.*—Age, usually forty to seventy; tongue coated; symptoms progressive and of short duration—a few months to a year; emaciation rapid; cachexia; repeated small hemorrhages, but not always; pain continuous, not paroxysmal, with no periods of relief, and not referring especially to the digestion period; anorexia, vomiting once or twice a day; later, coffee-grounds in vomitus; at times only occult blood in vomitus or stool; ectasia if the pylorus is involved; tenderness over the gastric region; no relief of pain by vomiting; anemia; free HCl diminished or absent; lactic acid present, also pus; sarcinæ rare and when present, few; Boas-Oppler bacilli in most cases; in some vomiting of blood (coffee-grounds), small in quantity and often repeated several times; melena slight. Leukocytosis moderate and eosinophilia present. Mucus present in some cases. Dysphagia and regurgitation if involvement is at cardia.

*Ulcer.*—Age usually twenty to forty; pain paroxysmal and worse after eating; remissions of pain, and relieved by vomiting; pain in epigastrium and in the back; local circumscribed tenderness in the epigastrium increased by pressure, at time slight tender point in the back; loss of weight; anxious expression of suffering; no cachexia; anemia; vomiting at height of digestion; appetite good; hyperchlorhydria usual, but not always; vomiting of blood in large quantity, at times of bright color; melena. Blood at times only occult.

*Benign Stenosis.*—Dilatation of stomach; peristaltic unrest and pain (spasmodic) preceding vomiting; long history; periods of improvement; hyperacidity usual; sarcinæ present in number; emaciation, but no cachexia; vomiting of large amount; usually no tumor, and if present very small and smooth on palpation; history of previous ulcer; usually no blood, as ulcer is practically healed or contracting; anemia present; no leukocytosis.

*Malignant Stenosis.*—Dilatation of stomach; peristaltic unrest; short history; no period of improvement; rapid loss of weight; pain continuous; cachexia; little or no free hydrochloric acid; lactic acid; Boas-Oppler bacilli; vomiting large amount and often odorous; at times coffee-grounds in vomit from ulceration of growth; anemia marked; leukocytosis.

*Chronic Gastritis.*—Long history of dyspepsia; absence of cachexia; no lactic acid; less anemia; no leukocytosis; intense pain absent, more feeling of discomfort; no real pain on palpation; mucus in gastric contents, HCl diminished or absent.

With nephritis and loss of cardiac compensation, or with *arteriosclerosis, especially the visceral type*, there may be *marked deficiency or even absence of free hydrochloric acid*, and these conditions must be held in consideration for the purpose of differential diagnosis.

*Achylia Gastrica.*—Total acidity much lower than in cancer, from 4+ to 2+ or less; free hydrochloric acid, pepsin and rennet absent; no mucus; scarcely any gastric juice; food particles coarse and nearly dry; course long; no cachexia; no lactic acid; may be considerable loss of weight. In transitional stage (commencing) some claim mucus is present.

Moreover, with pernicious anemia, we have achylia gastrica, but often other symptoms suggestive of carcinoma of the stomach, namely, pallor, weakness, loss of appetite, perhaps severe vomiting, even accompanied by blood, gas, and distress after eating. Examination of the blood shows the findings of pernicious anemia.

*Nervous Gastralgia.*—Patient nervous or hysteric; pain irregular or relieved by pressure; free intervals from pain; appetite variable; no regularity of vomiting; secretory function is variable; no tumor; no cachexia; character of food makes little difference as to symptoms.

*Carcinoma of the gall-bladder* is found in location of gall-bladder; follows respiratory movements of the liver; shows no lateral motility and does not allow expiratory fixation. Its position is unchanged if the stomach is inflated with air. It rarely causes dilatation of the stomach unless adhesions form; and dyspeptic disturbances are not marked, as a rule. Jaundice may be present. Head's zone is present.

*Enlarged lymph-glands* can hardly be mistaken, as there is the absence of gastric symptoms; no gastric findings, as in carcinoma of the stomach; and inflation of the latter organ enables a differentiation.

*Growths of the peritoneum or mesentery* are more diffuse and rarely movable on respiration, and gastric inflation and the symptoms enable one to differentiate.

*Exudates* or *adhesions* give none of the symptoms or gastric findings of carcinoma.

**Duration of Carcinoma of the Stomach.**—Osler reports 15 cases with fatality under three months; 45, under a year; 4 cases, two years or over; 1 case, two and a half years. The general average is about a year to a year and a half.

Cases involving the cardia or the pylorus are more rapid, as subnitrition occurs more quickly. The medullary type is more quickly fatal. Complications shorten the disease.

It is interesting to learn, however, that some cases of inoperable carcinoma of the stomach improve greatly after palliative gastroenterostomy. Eleven such cases have been collected from Czerny's[1] clinic, in which the patients were well from two to fourteen years later.

**Prognosis.**—This has been considered fatal, though recent results are more favorable. Surgery has relieved conditions temporarily and prolonged life, while medical treatment has failed, though it has helped to alleviate suffering.

[1] Wells, Resistance to Cancer, Jour. Amer. Med. Assoc., May 29, 1909.

Kocher has reported 1 case in which the patient was in good health five and a half years after resection of the pylorus for carcinoma; and Wölfler 1 in which the patient was well for five years when a metastasis occurred.

Recently a few isolated cases of apparent cure have been reported. In an analysis of the results of operative treatment of gastric cancer at Braun's Clinic at Göttingen, Creite[1] refers to 1 case in which fourteen years after resection of the pylorus for carcinoma the patient was in perfect health.

Recently, more favorable results have been reported. Leriche[2] has collected records of 89 patients on whom gastrectomy was performed, found in good health three years after operation; and of these 34 no less than five to ten years after operation.

Out of 79 cases treated by gastrectomy, Patterson[3] collected 33 (41.6 per cent.) who were free from recurrence three years or more after operation.

Deaver shows that we may expect 10 to 15 per cent. to be cured by radical operation.

According to Kausch,[4] Makkas traced 92 of Mikulicz's patients operated on before 1902, and found 17, or 14.3 per cent., well more than three years after operation. Further statistics are unnecessary. The radical operation, gastrectomy, evidently affords results in some cases.

Schlatter reported in 1897 the first successful case of total extirpation of the stomach, with survival of the patient for a considerable period. Bernays, of St. Louis, and others have reported the survival of cases for some time after operation. The operation has been generally abandoned and we find that the so-called *gastrectomies* are generally not complete removal.

**Treatment.**—There are two methods of treatment, surgical and medical, of which the only hope of cure lies in the former, medical treatment being only justificble if the case is inoperable or refuses operation, or as an adjunct to palliative operation.

*Surgery.*—Before referring to the radical or palliative methods in surgery, I desire to call to my readers' attention the necessity of the *education, not so much of the patient,* who will generally consent, if the matter is placed fairly before him, but *of the physician and the specialist in gastric diseases,* as to the value of *early exploratory laparotomy* for the *purpose of diagnosis.*

William Mayo[5] justly remarks that in an early exploratory incision we have the one diagnostic resource which is reliable, and which must be resorted to in a large majority of cases before a surgical diagnosis can be made, and without it the truth is but slowly established at the expense of progressive hopeless involvement. It can be safely accomplished through a small incision. He further calls to our attention that the chemic findings of the gastric secretion gain in diagnostic importance with the progress of the disease and become of the greatest value when the patient is *in a hopeless condition*, and that exploration should not be delayed by reason of the inconclusive nature of the results. He has further demonstrated that about 60 per cent. of cases of cancer begin in the pylorus and 70 per cent. in the pyloric region, and that the early diagnosis of cancer depends in a great measure upon the introduction of the *mechanic phenomena from obstruction at the pylorus*. It is the interference with gastric motility which first calls the patient's attention to his trouble and not the presence of the cancer itself. Moreover, a case with marked symptoms of cancer of the stomach, but without any evidence of pyloric obstruction, proves, on exploration, to be the victim of advanced and hopeless disease of the body of the organ, in which there were no symptoms during the operable period. The presence of a tumor does not demonstrate inoperability, as a small movable tumor in the pyloric region may be a favorable indication. Limita-

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[1] Jour. Amer. Med. Assoc., Aug. 24, 1907, p. 723.

[2] Revue de Medecin, Jan., 1907.

[3] Internat. Med. Annual, 1908, p. 537.

[4] Ibid.

[5] Ann. of Surg., March, 1904.

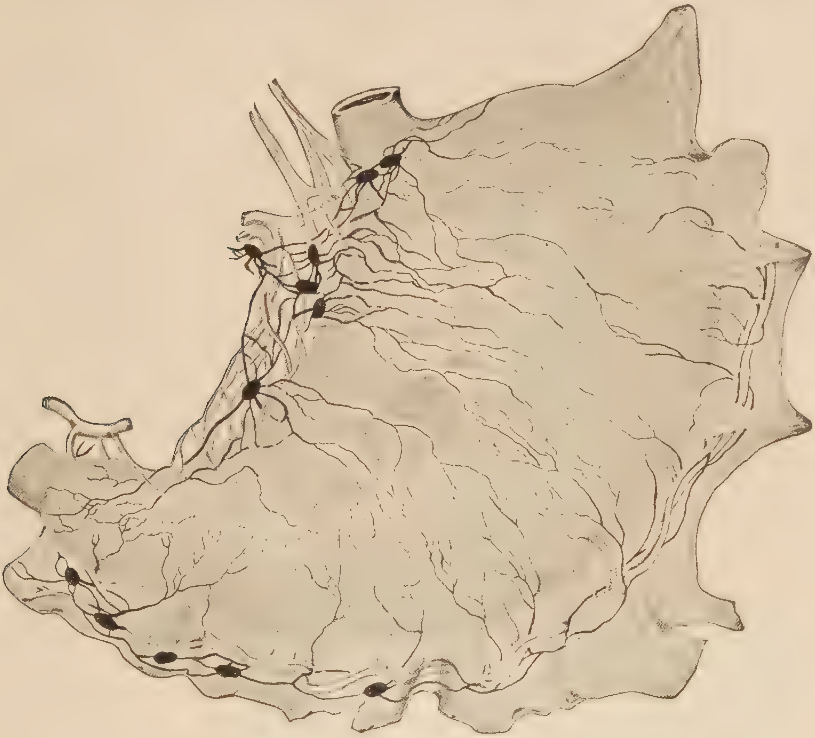

Fig. 167.—The lymphatics of the stomach (Moynihan, after Cunéo).

tion to the pyloric end and mobility are the important factors, also the degree of lymphatic infection.

*Pyloric Stenosis is a Surgical Disease Whether it is Benign or Malignant.*—It seems, therefore, especially sound doctrine in all cases with symptoms pointing to pyloric stenosis to perform exploratory laparotomy to settle the type of stenosis and immediately further operation, of greater or lesser extent, the character depending on the cause of obstruction. This rule, the author believes, should be followed out, whether the patient be over forty years of age or not. *Pyloric stenosis, in any event, is a surgical condition.*

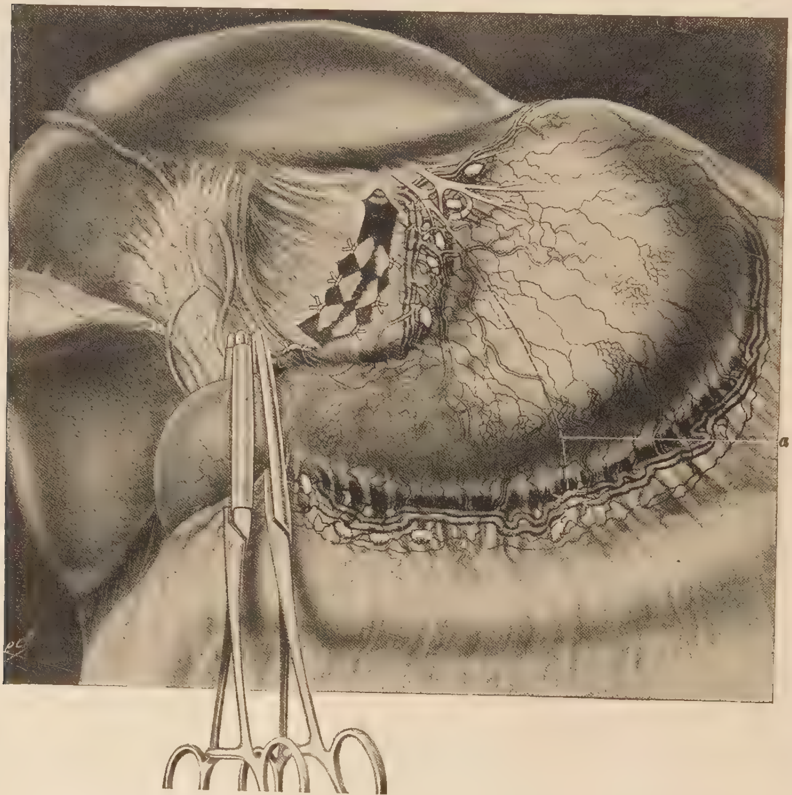

Fig. 168.—W. J. Mayo's method of partial gastrectomy for cancer of the stomach. Ligation of gastrohepatic omentum and superior vessels in such manner as to leave all the lymph-nodes attached to the part of the stomach to be excised; also lines of division of duodenum and stomach: *a*, Mikulicz-Hartmann's line (Fowler).

W. L. Rodman[1] also advances numerous legitimate reasons for exploratory laparotomy. Parker Syms[2] refers to the necessity of operation in chronic gastric ulcer, in the precancerous stage, and also to the value of exploratory laparotomy in all doubtful cases to settle the diagnosis. Rodman holds that *rapid emaciation* with *gastric symptoms in a person over forty years of age almost invariably calls for an exploratory laparotomy.*

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[1] Jour. Amer. Med. Assoc., Jan. 18, 1908, pp. 165–169.

[2] N. Y. Med. Jour., July 16, 1910.

In elderly persons, whether previously in good health or not, with *gastric symptoms, anemia, and rapidly developing emaciation,* after frequent examinations, both of the patient and gastric contents for several weeks, even if no definite results are secured by analysis and no tumor be detectable, *exploratory laparotomy is indicated.* This is true whether symptoms of stenosis are present or not. Some patients, of course, will not consent to any operation until even palliative operative procedure is too late.

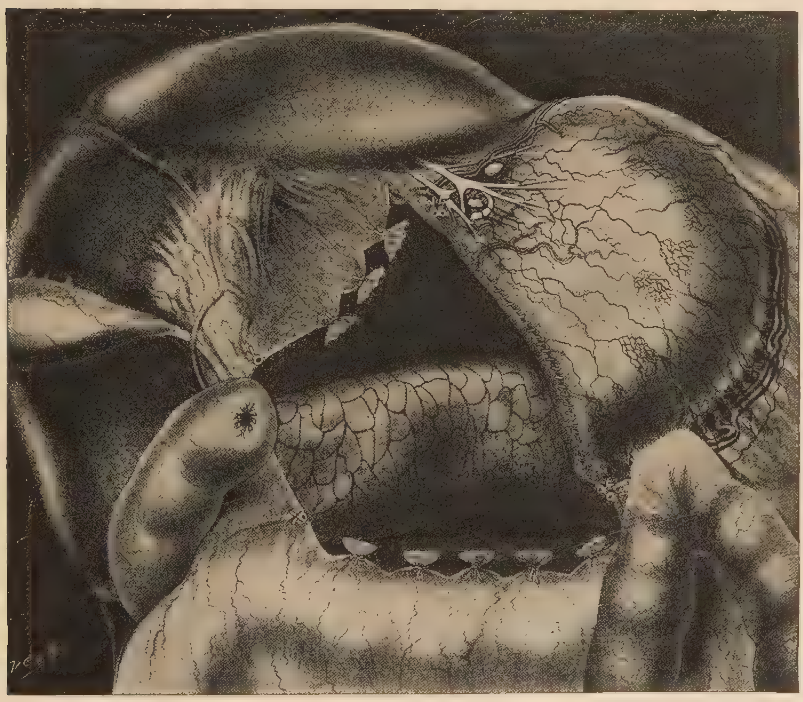

Fig. 169.—W. J. Mayo's method of partial gastrectomy for cancer of the stomach. Operation completed (Fowler).

I have seen a number of abdomens opened and immediately closed as inoperable, in one case notably the entire stomach wall being infiltrated. If the medical profession would recognize the value of exploratory incision, I believe many lives could be saved.

*Radical Operation.*—Billroth, in 1878, was the first to prove the possibility of resection of the pylorus for cancer, but it has been clearly demonstrated that this operation is insufficient. Mikulicz has pointed out that on the lesser curvature the blood and lymph-vessels lie in the wall of the stomach itself, and that it is necessary in every case of pyloric cancer to remove all the lesser curvature to the gastric artery. Cuneo has demonstrated that there are but few lymph-glands along the greater curvature and that these are chiefly confined to the pyloric region.

One must remember in operating that carcinomatous emboli in the early stages do not necessarily involve glands one after another, but may primarily pass into glands at a considerable distance.

The illustrations (Figs. 167–169) demonstrate the glandular relations, the correct line of incision, and the completed operation.

In all cases of pyloric cancer a partial gastrectomy and pylorectomy should be performed. If cancerous metastases or marked adhesions are present, radical operation is contraindicated, as it is in the case of extreme debility or old age.

C. H. and W. J. Mayo's latest statistics for this operation, from April, 1897, to Janurary 27, 1910, are 34 deaths out of 266 operations, and show 12.4 per cent. mortality, and Deaver's, 11.11 per cent. The latter has tabulated 393 cases by various operators, with an average 26.5 per cent. fatality.

Out of the Mayos[1] 266 operations, 42 were for benign tumors or ulcers, or where the diagnosis was not microscopically established. They are, therefore, excluded from the following statistics.

*Table 1.*—Operations for carcinoma involving the pyloric end of the stomach:

| | |
|---|---|
| Total number | 224 |
| Males | 163 |
| Females | 61 |
| Age of oldest | 81 |
| Age of youngest | 30 |
| Average age | 55 |

*Table 2.*—Patients operated on over five years ago:

| | |
|---|---|
| Total number | 50 |
| Present condition known | 39 |

Alive and well (1, eight years two and a half months; 1, eight years;[2] 1, seven years two months; 1, six years eleven months; 1, six years; 1, five years three and a half months; 1, five years)—6 still alive and well.

*Table 3.*—Patients operated on over four years ago:

| | |
|---|---|
| Total number | 85 |
| Present condition known | 64 |
| Alive and well | 13 |

*Table 4.*—Patients operated on over three years ago:

| | |
|---|---|
| Total number | 117 |
| Present condition known | 88 |
| Alive and well | 18 |

There are 107 patients operated on less than three years ago, too recent for statistical value. The Mayos advocate partial gastrectomy with pylorectomy when possible.

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[1] Partial Gastrectomy (Mayo), Jour. Amer. Med. Assoc., May 14, 1910.

[2] One has died of recurrence.

*Palliative Operation.*—This should be performed when radical operation cannot be carried out, to prevent death from starvation and to remove the irritating effect of food with the resulting pain. The operations are:

(*a*) *Gastrostomy* is the formation of a fistulous opening into the stomach; indicated in cancerous stenosis of the cardia, with rapid loss of weight and severe pain.

(*b*) *Gastro-enterostomy* is an anastomosis between the stomach and small intestine, in cancer of the pylorus with stenosis; for similar reasons.

These operations prolong life and give the patient considerable comfort, and there is often temporary increase in weight. They are less severe than the radical operations, and are only contraindicated in extreme debility or great age, with the patient in such condition that fatality would be assured. Gastrostomy can be performed under cocain (local) anesthesia if necessary. I have already referred to the fact that in 11 of Czerny's cases the patients were living from three to fourteen years after gastro-enterostomy.

**Medical Treatment.**—*Diet.*—The first important feature is the endeavor to relieve the condition of subnutrition and to give the patient food which he will most easily assimilate. If there is stricture of the pylorus or esophagus, it will necessitate the use of liquid food entirely, or mushes in addition, if there is less severe obstruction.

If the cancer involve the body of the stomach alone, food of more solid character can be taken, as the motor functions are not as greatly interfered with. In the dietary we include milk, koumiss, matzoon, bacillac, farinaceous food; soups, with finely divided vegetables, such as pea, bean, and potato; broths, gruels, bouillon; raw or soft-boiled eggs; butter in plenty, tea, weak coffee, and cream; crackers softened in water and milk-toast.

In some cases, chicken, squab, scraped meat, sweetbread, stale bread, oysters, fish, etc. Sanatogen (flavored), given in 2-dram (8.0) doses, up to 1 ounce a day; somatose, Wyeth's beef-juice, Mosquera's beef-jelly, soluble beef peptonoids, Armour's extract of beef, tropon, and rare beef-juice, are all of value.

I have employed as many as 8 raw eggs a day beaten up in milk, in addition to other foods. They possess great nutritive value. Russell's emulsion of mixed fats is of service. Food should be administered in divided small meals, four to eight a day. The calorie value can be estimated, but the chief criterion is their digestibility and the increase in the patient's weight, which should be carefully recorded. Temporary increase in weight can often be secured.

It is quite remarkable what results one can secure by proper methods of feeding. One case who had lost 70 pounds in weight, the weight on admission to the Red Cross Hospital being only 120 pounds, was vomiting all food, emesis occurring seven or eight times daily. The patient's pulse was hardly detectable, and he was so feeble that he could only walk when supported. There was a marked growth at the pylorus, and the lower border of the stomach lay three finger-breadths below the umbilicus. Though palliative gastro-enterostomy was indicated, the writer believed that the patient's physical condition precluded an immediate operation, and in this opinion the surgeons concurred. Lavage twice daily was immediately begun, the patient being fed on each occasion through the stomach-tube, with additional feedings, and nutritive enemata. For example, 1 quart (liter) of peptonized milk, 6 to 8 raw eggs, sanatogen, 1 ounce, iron tropon, 3 drams, were given in divided doses by mouth, with the addition of broths and strained gruels—an extra pint in divided doses. Butter was added to these, and later crackers crumbled in the broths and thicker gruels. Four nutritive enemata, each containing 3 ounces of peptonized milk and 1 raw egg, were given daily. Rose's belt was applied to elevate the stomach, and after each feeding the patient was turned on his right side and kept in this position for half an hour to facilitate emptying the stomach. Olive oil, 2 ounces, were given, A. M. and P. M., to facilitate the passage of food through the pylorus and also aid nutrition, and belladonna tincture, 10 minims t. i. d., to relax spasm. Vomiting only occurred twice after treatment was begun. The patient gained 20 pounds in eight weeks and then underwent a successful gastro-enterostomy, living a year in comfort.

*X-rays.*—There have been various claims made for the value of the *x*-ray in the treatment of internal cancer, such as of the stomach or intestines, that it diminishes the size of the growth and relieves pain. I have seen cases in which the pain seemed to be somewhat relieved, but never any permanent results. In the treatment of skin cancer definite or even curative results have been secured. Beck's eventration treatment with employment of the *x*-rays is described at the end of the chapter. Incidentally Morton has recommended the use of fluorescent media internally in connection with the *x*-rays, but Henry Piffard and S. Tousey have conclusively exploded his theory and shown that fluorescence does not occur. Direct light rays are necessary.

The first researches with the internal administration and dosage of fluorescein were reported by me in connection with *gastrodiaphany.*[1]

*Radium.*—The radium treatment for cancer of the stomach and esophagus was first introduced by Einhorn.[2] He first employed for the stomach a hard-rubber capsule that can be unscrewed and which contains a glass radium flask (Curie 20,000 strength). To the rubber capsule is attached a silk thread, in which several knots are tied, indicating the distance from the lips to the cardia and how far the capsule lies from the cardia. The capsule is introduced like his stomach-bucket. The thread is tied to the lobe of the ear and the capsule left in the stomach for one hour or more. He has recently devised improved instruments.[1] One type, like that depicted in Fig. 170, has in addition a small canal at the margin of the capsule, which can be threaded on a guide. A duodenal buckle with thread is swallowed and the capsule is later passed along the thread to the pylorus. His radium introducer consists of a flexible introducer and mandril, which are withdrawn after placing the capsule in the desired position.

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[1] Med. News, April 10, 1904.

[2] Med. Rec., March 5, 1904.

Einhorn states he has not yet formed definite conclusions as to results, but it seems to be of palliative benefit. Serious burns have resulted from prolonged exposure to radium, and no definite results are yet reported. I *would, therefore, not recommend the method.*

An instrument that is connected to a thin rubber tube in which a mandrin is slipped for the purpose of introduction, and then the latter removed, has been devised by Einhorn for the treatment of malignant esophageal stricture, but his most recent instruments are depicted in Fig. 170.

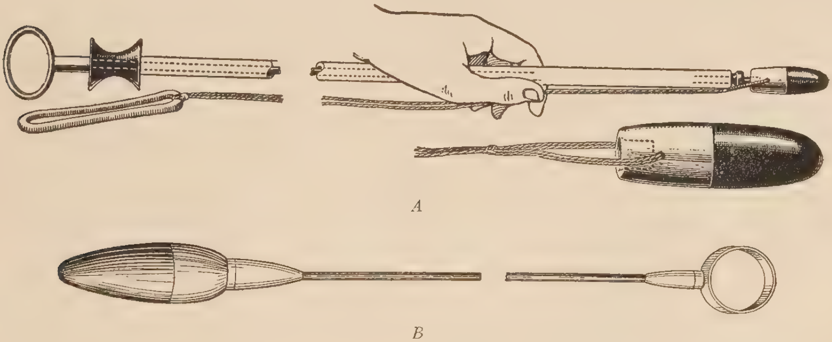

Fig. 170.—*A*, Radium introducer; *B*, radium receptacle for stomach and esophagus, with whalebone stem.

He recommends leaving it in the esophagus from half an hour to an hour, and claims an increase in the permeability of the stricture, less pain, and increased ability in swallowing. As in this case the radium can be directly applied and some definite results have been secured, the method might be of value. The radium introducer is the best instrument. The question of damage from overexposure *must be carefully considered.* One case reported, of six hours' exposure, the writer believes highly dangerous. The method is evidently worthy of further investigation. *It is only applicable to patients refusing operation.*

**Drugs.**—*Sodium Iodid.*—In stricture of the cardia, Boas has recommended sodium iodid, 30 to 45 grains (2.0–3.0 grams), in divided doses during the day, and claims that during a treatment of over six months the patient gained a little in weight and was relieved from some of the symptoms. Even though transient improvement occurs, it is worthy of trial if it adds to the patient's comfort. Administration for a brief period would decide the latter question.

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[1] Med. Rec., Sept. 23, 1911.

*Thiosinamin.*—Sachs[1] claims to have been successful with thiosinamin by hypodermic use in the treatment of 2 cases of pyloric stricture; and Michaelis[2] found it softened an esophageal stricture and enabled him to dilate it with bougies.

Thiosinamin (fibrolysin) is moderately soluble in water, soluble in 3 parts of alcohol, and readily soluble in ether. Hypodermic injection of fibrolysin can be given in 15 per cent. alcoholic, or 10 per cent. glycerin, solution.

The average dose is ½ to 1½ grains (0.032–0.1 gram). Einhorn advises its use by mouth.

It is worthy of trial in stenosis of the esophagus or pylorus. The following is useful:

| | | |
|---|---|---|
| ℞. | Thiosinamin | 0.5 (7½ gr.); |
| | Glycerini | 6.0 (ʒiss); |
| | Syrup. cort. aurant | 20.0 (ʒv); |
| | Aq. destil. q. s. ad. | 60.0 (℥ij).—M. |

Sig.—Teaspoonful in water t. i. d.

*Arsenic* has been recommended, Fowler's solution, 3 to 5 drops (0.2–0.3) daily; or sodium arsenate, $\frac{1}{30}$ to $\frac{1}{25}$ grain (0.002–0.0026) t. i. d., but their chief value is combined with iron, such as Gude's peptomangan, or iron tropon, to combat the anemia.

| | | |
|---|---|---|
| ℞. | Blaud's iron pill (fresh) | gr. v (0.32); |
| | Sod. arsen | gr. $\frac{1}{50}$ (0.0013).—M. |

One pill, made soft with honey, is an excellent combination, given t. i. d.

*Condurango* was first recommended by Friedreich in 1874 for the treatment of cancer, but it has no specific action.

Alone or combined with dilute hydrochloric acid (suggested by Ewald), it is an excellent stomachic to improve the appetite, and at times increase of weight may result.

It may be given as the fluidextract of condurango, 15 to 20 drops (1.0–1.3) in water t. i. d., with or without dilute hydrochloric acid, half an hour before meals; or the decoction of condurango may be employed:

| | | |
|---|---|---|
| ℞. | Decoct. condurango | 20.0 to 25.0 gram.; |
| | Water | 200.0 to 250.0 c.c.—M. |

Tablespoonful t. i. d. before meals.

Other stomachics, such as are advised in chronic gastritis, are of service:

| | | |
|---|---|---|
| ℞. | Acidi hydroch., dilute | ʒij (8.0); |
| | Tr. nuc. vomic | ʒij (8.0); |
| | Comp. tinct. cinchona | ℥ss (16.0); |
| | Aq. destil. q. s. | ℥iv (125.0).—M. |

Dose, one to two teaspoonfuls in water t. i. d. before meals.

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[1] Ther. d. Gegenw., 1907, No. 1.

[2] Med. Klin., 1907, No. 10.

*Methylene-blue.*—Einhorn[1] has employed methylene-blue in capsules once or twice daily for some years past—3 grains (0.2 grams). In 1 case the tumor appeared smaller for a time and did not increase in size; for eight months the patient was free from pain and lost no further weight. Later it grew again and the case succumbed. He believes it exercises a beneficial action in some cases.

A. Jacobi[2] advocates its use in inoperable intra-abdominal cancer, having used it for about fifteen years. He reports cases of various types and claims mitigation of symptoms, prolongation of life for some years in several cases, and a temporary retrogression of the tumor. He does not claim to have absolutely cured a case. He believes that exposure to sunlight is an aid, as methylene-blue is fluorescent.

He advocates doses (divided), commencing at 2 grains a day and increasing to 6 grains, combining belladonna, and suggests the following:

| | | |
|---|---|---|
| ℞. | Methylene-blue | gr. vj (0.4); |
| | Ext. belladonna | gr. $\frac{3}{4}$ (0.048); |
| | Arsen. acid | gr. $\frac{1}{10}$ (0.0065).—M. |

Divide into four pills.

Sig.—One t. i. d. after eating and at bedtime.

*Methylene-blue* treatment may be tried in inoperable cases as a *palliative.*

*Trypsin Treatment.*—For the theory of Beard, on which he bases his so-called trypsin treatment, I refer my readers to his various articles. There have been cases of apparent cures reported, and others of complete failure.

Rice[3] reports a case of supposed cancer of the larynx with cure; while Wm. S. Bainbridge[4] demonstrated a reported cure by Morton to be *an absolute failure.*

Shaw-Mackenzie[5] and Carter[6] report cures in apparently hopeless cases, while Ball and Thomas[7] report unfavorably in 11 cases. There are many other reports for and against its use. Cleaves believes this treatment to be justified, noting a relief of pain, an improvement in metabolism, and improved mental condition. She described a tryptoglycogenic reaction as occurring from its use, consisting of a moderate leukocytosis and eosinophilia.

Trypsin treatment of cancer is a *failure.*

Wm. S. Bainbridge, of New York, has had under observation about 100 cases undergoing this method of treatment, which are reported in the Medical Record of July 17, 1909.

[1] Deutsch. med. Wochenschr., 1891, No. 18.

[2] Jour. Amer. Med. Assoc., Nov. 10, 1906.

[3] Med. Rec., Nov. 24, 1906.

[4] N. Y. Med. Jour., March 2, 1907.

[5] Med. Press, May 15, 1907.

[6] Ibid., March 18, 1907.

[7] Archiv. Middlesex Hosp., vol. ix, in Prac., August, 1907.

Space will only allow me to insert a small portion of his summary, in which he deduced the following:

1. The internal medication with holadin and ox-gall aids digestion and increases elimination.
2. Aiding digestion, increasing elimination by skin, kidneys, and bowels, and decreasing local absorption are the most important features of the treatment.
3. The régime, by increasing resistance, may in some cases decrease the rapidity of the malignant process.
4. Control cases given sterile water plus the régime did as well as those on the full enzyme treatment.
5. Trypsin injection seems in some cases to cause disintegration (liquefy) of the cancerous tissue in the center of the mass, but the periphery continues to grow.
6. Amylopsin injection seems to diminish cachexia in some cases.
7. In a small percentage of cases hemoglobin improves under trypsin injection.
8. A steady increase (6 to 12 per cent.) in the eosinophiles occurred in most cases during the trypsin injections, which lessens under amylopsin injection. There was no increase when they were given by mouth.
9. The claims for eosinophilia as a test were not substantiated.
10. Beard's enzyme treatment does not check the cancerous process.
11. It does not prevent metastases.
12. *It does not cure cancer.*

The author *would state positively* that the *trypsin treatment for the cure of cancer is absolutely without value.* He has followed up several cases on which he reported in his former edition of this volume, and *all have died* of the cancerous condition, the growths having later rapidly increased in size. In some of the cases the mental condition of the patient seemed improved somewhat, and in 1 case there was apparently relief of pain and some diminution in the size of the tumor, which later increased in size. The *method is useless*, and, therefore, is not described in this edition.

*Injections of Cancer Residue.*—J. Walter Vaughan, at the annual meeting of the Michigan State Medical Society,[1] Sept. 28–29, 1910, described his experiences with injecting the non-toxic protein of the cancer cell, or cancer residue, in the endeavor to find proof with regard to the formation of a specific ferment by means of a study of the various blood elements, particularly the leukocytes. Vaughan experimented on 12 persons suffering from malignant growth. It *is too soon* to draw conclusions from *this work at the present time.* The injection of the cancer residue has, at any rate, apparently had the effect of relieving the pain attendant on cancer.

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[1] Jour. of Michigan State Med. Soc.; also N. Y. Med. Jour., Oct. 15, 1910.

*Treatment of Carcinoma with the Body Fluids of a Recovered Case.*—The late E. Hodenpyl[1] reports a rare case of recovery from extensive carcinoma with residual chyliform ascites. Mice who had developed strains of cancer cells were injected with varying amounts of this ascitic fluid. The results were a marked necrosis of the tumors, a diminution in their size, or their complete disappearance. Injections of this ascitic fluid were then made in small quantities into human beings in cases of carcinoma of various types. Hodenpyl reports that in all cases the tumors have grown smaller, in some they have disappeared altogether, in others there was necrosis of the tumor tissue with the subsequent formation of connective tissue. He further stated there was no reaction after the injections in any tissues other than the tumor, nor were there any systemic effects manifest even after large venous infusions. Hodenpyl does not announce a new cure for cancer, but merely the effects noted. R. Weil investigated the "Properties of Ascitic Fluids, Especially in Cases of Cancer,"[2] among his experiments employing Hodenpyl's fluid. The results were almost entirely negative in character, that is to say, they have not indicated the existence of any specific character such as would serve to differentiate with certainty fluids derived from cancerous individuals from those of another origin. The writer has further made inquiries as to the results of the injections of this peritoneal fluid in about 20 cases, and finds that though in some there was temporary improvement, ultimately the carcinomatous condition *progressed*. There is evidently no curative value in the method. Risley's conclusions from his experience at the Massachusetts General Hospital confirm this view.[3]

*Cancer Vaccine (Neoformans).*—This has been recommended for injection. There have been reports of temporary improvement, relief of pain, and of temporary diminution in the size of the growth. I can find *no authentic record of cure by its use.* C. A. Aaron[4] reports temporary improvement from its use in a case of carcinoma of the splenic flexure of the colon. From 50,000,000 to 150,000,000 by hypodermic injection, at intervals of several days for five or six injections, and then an interval, apparently gave the best results.

Doyen first advocated the method and Karwacki[5] reports his conclusions. I can see no objection to its use in inoperable cases, or as an adjunct with palliative operative procedures. I believe it useless as to cure, but it has apparently been of temporary assistance in some cases in promoting the comfort of the patient.

*Thyroid Extract.*—C. G. Am. Ende[6] reports some apparently antitoxic properties in the fluidextract of the fresh thyroid gland to cancerous growth. There was some property of neutralizing cancer toxins. The bodily and mental vigor and the almost complete prostration of advanced carcinomatosis were relieved. Later the *x*-ray was added to the treatment. Better results were secured by the addition of the thyroid extract to the *x*-ray local treatment, than by the latter alone. Prolongation of life, with increased comfort, occurred in several cases. There was *no cancer cure.*

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[1] Med. Rec., Feb. 26, 1910.

[2] Jour. of Med. Res., vol. xxiii, No. 1 (New Series, vol. xviii, No. 1, pp. 85–94, Aug., 1910).

[3] Jour. Amer. Med. Assoc., May 13, 1911.

[4] Med. Rec., Oct. 1, 1910.

[5] Warschauer med. Wochenschr., 1906, No. 5.

[6] A Contribution to the Treatment of Cancers, Med. Times, Sept., 1909.

*Thymus.*—F. Gwyer[1] reports the use of dried and powdered thymus of the calf in doses of 1 to 2 drams (4.0–8.0) three or four times a day. He claims *diminution* of pain; reduction of size of growth, improved digestion, and diminution or arrest of the growth. Elimination is through the secretory organs, and considerable reaction occurs, so it cannot be used for over three wekes. Gwyer has also experimented with thymus with arterioselerosis and diabetes, rheumatoid arthritis, hemorrhoids, disease of the gall-bladder, and pulmonary tuberculosis. There is a certain amount of danger from auto-intoxication and the method is still under experiment, so that I would not advise its employment.

*Coley's fluid* (*toxins of the streptococcus* of erysipelas and of *Bacillus prodigiosus*) might be tried, though the results have been more favorable in inoperable sarcoma and rarely in carcinoma. Coley *recommends* its *use, particularly after operation for carcinoma to lessen the chance of recurrence.*[2] The injection should be begun with ¼ minim diluted with sterile water to ensure accuracy of dosage, given by hypodermic. Daily injections should be given, increasing by ¼ minim, until the desired reaction and temperature of 102° to 104° F. has been obtained. The dose should then be no longer increased until it fails to give a reaction, when it can again be increased from ¼ to ½ minim. The largest dose has been 7 to 8 minims. Duration of treatment from six weeks to four to six months. In the cases of inoperable sarcomata from 30 to 80 injections were given. The best toxins are prepared by Tracy, pathologist to the Huntington Cancer Research Fund. The method is worth trying in inoperable carcinoma.

Bier has reported some improvement in superficial cancer by injection of heterologous blood of a pig (10 to 20 c.c. at a dose); and Leyden and Bergel have experimented on animals suffering from cancer with the injection of liver extracts, but these methods are also experimental.

*Vomiting and Ectasia.*—Systematic lavage is indicated for these conditions with normal saline solution or milk of magnesia, 1 to 2 ounces (30.0–60.0) to the quart (liter) of water; if marked fermentation, resorcin, 15 grains (1.0), can be added; or glycothymolin or listerin, 1 dram to 1 pint (4.0–500 c.c.) of water, etc.

*Hemorrhage.*—This is usually not severe. Morphin, ¼ grain

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[1] Ann. of Surg., July, 1907; Ibid., April, 1908; N. Y. Med. Jour., Feb. 19, 1910.
[2] Trans. of New Hampshire Med. Soc., May 12 and 13, 1910.

(0.016), by hypodermic; the ice-bag; gelatin, 5 to 10 per cent. solution, by mouth, 2 drams to ½ ounce (8.0–16.0), every one to two hours for twelve hours are useful; also hypodermics of ernutin, 5 minims (0.296 c.c.), or ergot fluidextract, and the methods described for hemorrhage under Gastric Ulcer.

*Trémolière's Solution.*—Gelatin (5 per cent. solution) with calcium chlorid (2 per cent.) therein. Dose, ½ to 1 ounce (30.0–60.0) by mouth, repeated every four hours, is of value. There are not the indications to relieve hyperchlorhydria as in ulcer, so rectal feeding may be instituted for twenty-four hours or longer. The ice-bag should be kept on for twenty-four hours. Gelatin thereafter (3 per cent. solution) should be used for several days, in divided doses up to 12 ounces (375 c.c.) in twenty-four hours. White of egg is of value on the following day, and then fluid diet, and a gradual return to the usual feeding. Lactate calcium, 10 grains (0.6), is preferable to calcium chloride. It can be given t. i. d.

*Pain.*—The application of heat by the hot-water bag or hot poultices *is indicated.* Boas recommends 3 to 5 drops of chloroform on ice to be given occasionally. Chloral hydrate, 3 to 5 grains (0.2–0.3) in water, has been recommended by Ewald, but if the patient be very weak it is a dangerous remedy. Lavage will often relieve acute attacks, especially if stenosis with dilatation are present.

Orthoform or, preferably, orthoform hydrochlorid, which is more soluble in water, can be given t. i. d., 5 to 7½ grains (0.3–0.5). The same dose of anesthesin is at times of service.

Tincture belladonna, 10 minims (0.66) t. i. d., or extract belladonna, ½ grain (0.022) t. i. d., are valuable. Heat, belladonna, lavage, and orthoform or anesthesin should first be tried. If they fail, it may be necessary to employ codein, preferably, ⅛ to ½ grain (0.008—0.032) by mouth, or ¼ grain (0.016) by hypodermic; or, as a last resort, morphin, ⅛ to ¼ grain (0.008–0.016). In terminal cases the opiates are indicated.

*Bowels.*—For *constipation,* enemata of soap and water, with or without olive oil, bowel irrigation, injection of olive oil (8 ounces to 1 pint (250–500 c.c.) at night, to be retained, rhubarb pills, the cascara preparations, aloin and belladonna compound pills, purgen or phenolax tablets, regulin, and compound licorice powder, are all of service.

For *diarrhea,* the bismuth preparations, subnitrate of bismuth, 1.0 to 2.0 (15 grains to ½ dram), several times a day; or bismuth salicylate, 5 to 10 grains (0.32–0.64), three or four times a day; or subgallate of bismuth, the same dose; or subcarbonate bismuth, 1.0 (15 grains), three or four times a day, are of value. Chalk mixture or compound tincture catechu or kino, in ½-dram (2.0) doses, may be combined; or at times tincture opii, 10 minims (0.892), or tincture opii camphorata, 15 to 30 minims (0.888–1.7776 c.c.), may be required in addition.

*Stenosis of the Cardia.*—This may require cautious dilatation, preferably with soft tubes, if possible. Palliative gastrostomy is usually required.

In conclusion, I desire to refer to an interesting communication by Carl Beck.[1] In several cases of intra-abdominal cancer, one, notably of the pylorus, incision was made over the growth, and the latter stitched to the skin, making it practically cutaneous. The *x*-rays were then applied, with the apparent disappearance of the growth in several cases. In some, the wounds were allowed to granulate, and in others the stomach was reduced after separation of adhesions. In some of the cases the report was certainly favorable. It would seem that in cases of cancer of the stomach in which extirpation is impossible, gastro-enterostomy combined with Beck's treatment, just described, and forced feeding to improve the resisting power of the patient, might give the best results.

## OTHER TUMORS OF THE STOMACH

*Multiple Polypi of the Stomach Undergoing Malignant Changes.*—An interesting case[2] of this type has been reported associated with multiple subcutaneous hemangio-endothelioma, multiple lymphangio-endothelioma of the intestines, general vascular sclerosis, and cirrhosis of the liver. Vomiting, dropsy, and ascites were prominent symptoms.

Other tumors of the stomach (excepting carcinoma) are comparatively rare. Sarcomata, lipomatoma, fibromata, and myomata have been found, and also polypoid excrescences due to proliferation of the glands.

**Sarcoma** is the most common of these varieties, and may be primary or secondary.

Primary myosarcoma and fibrosarcoma are generally in the form of circumscribed nodules in the stomach wall, while lymphosarcoma is flatter and infiltrates.

The tumors vary in size and form, and are situated generally on the greater curvature. Metastases are frequent.

Primary lymphosarcoma appears most frequently between twenty and thirty-five years of age, while the other types occur more frequently in older subjects. It has occurred at the age of three and a half years. The cases are about equally divided as to *sex.* Corner[3] and Fairbank report 58 cases.

*Symptoms of Sarcoma of the Stomach.*—These appear somewhat insidiously, in some cases gradual emaciation being first noted; the stomach symptoms are practically the same as in carcinoma: loss of appetite, sour belching, a feeling of pressure and fulness, disagreeable taste, pain, vomiting, and, finally, coffee-grounds in the vomitus. Dilatation of the stomach, if pyloric involvement and tetany have been observed in this type of case. Some of the cases run a rapid course, with marked anemia, with an absence of hematemesis and no gastric dilatation, though the tumor grows rapidly in size.

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[1] On External Röntgen Treatment of Internal Structures (Eventration Treatment, New York Med. Jour., March 27, 1909.)

[2] Bull. of Johns Hopkins Hospital, July, 1910.

[3] Practitioner, 1904, lxxii, 810.

*Gastric Analysis.*—Absence of free hydrochloric acid; presence of lactic acid; Boas-Oppler bacilli are frequently present.

In effect, we may say the gastric findings and symptoms are similar to carcinoma.

The methods of physical examination are the same as in carcinoma of the stomach.

**Average duration** is from one to one and a half years. Schlesinger and Kundrat have shown that certain factors may be utilized for the purpose of differential diagnosis.

Metastases of the skin are more frequent with sarcoma, and excision, with examination of a cutaneous nodule, when such is present, will afford positive information. Metastases in the intestines occur more frequently with lymphosarcoma, and these do not produce stricture, but dilatation; while carcinoma causes stenosis of the gut.

The lymph-glands are more swollen in sarcoma.

The *spleen* is also swollen in sarcoma, not so in carcinoma.

The tongue follicles are at times swollen and tumefied and infiltration of the tongue may occur; there is a symmetric arrangement of the ridges, nodules, and papillæ.

Harlow Brooks believes the growth usually appears at the lesser curvature of the stomach, though it may occur in other regions. It may be multiple.

**Treatment.**—Early surgical operation, if possible, the indication being the same as in carcinoma.

Lavage is indicated if dilatation be present. Coley's fluid (erysipelas toxin) has proved of value, especially in some cases of sarcoma, and should be tried in inoperable cases. Severe reaction and renal disturbance may follow its use, so it should be employed with caution.

The diet should be the same as in carcinoma, and the use of stomachics.

Arsenic (Fowler's solution), beginning at 5 minims (0.296) and gradually increasing to 15 minims (0.888), t. i. d., can be tried in lymphosarcoma.

*Benign Tumors.*—Benign tumors of the stomach are a rarity and practically impossible to differentiate in many cases. Polypi are the most common, though fibroma, lipoma, and myoma have occurred. The cachexia and gastric findings of malignancy would be absent.

With benign pyloric stenosis, a tumor (thickening) is palpable in some cases, but the symptoms would be of benign stenosis, with dilatation of the stomach, cachexia absent, and usually hyperacid gastric contents.

Occasionally a small tumor has been noted lying on the greater curvature, due to enlargement of a lymph-gland secondary to an inflamed ulcer.

## APPARENT TUMORS OF THE STOMACH

Einhorn[1] has described cases of apparent tumors of the abdomen which have been mistaken for tumors of the stomach, notably, prolapse of the left lobe of the liver, exposure of the aorta (abdominal), thickening of the abdominal muscles (recti), and adhesions of the lesser curvature. Enteroptosis of a considerable degree is usually associated with the first two conditions and the history is a long one. Pulsation of the aorta may be mistaken for aneurysm. Cirrhosis of left lobe of liver may simulate cancer of the stomach on palpation. The gastric analysis and other symptoms will differentiate these conditions. With gastroptosis the pancreas has been palpated and mistaken for gastric tumor. In some cases a transient tumor in the region of the stomach, dependent evidently on spasm, as from ulcer, has been noted. Schnitzler[2] observed such a case.

Thickening of the recti is diagnosed as follows: The thighs and knees are well flexed, and the head and shoulders elevated, so as to produce marked relaxation of the abdominal muscles. It will then be possible to slip the finger-tips beneath the edges of the relaxed and thickened rectus muscle. With adhesion, pain on distention of the stomach, motor disturbances, etc., are present. (See Perigastritis.)

## FOREIGN BODIES IN THE STOMACH

These may be swallowed accidentally or purposely, or may be gradually deposited in the stomach.

Among such articles are pins, needles, scarf-pins, knives, spoons, forks, artificial teeth, glass, hooks, pens, buttons, balls of hair, bits of iron, nails, lead, wood, and even the stomach-tube. Lunatics, idiots, and young children frequently swallow foreign bodies, as do trick knife-swallowers. Cases have been reported of patients who have worked with an alcoholic solution of shellac, and who had swallowed small quantities daily, with the ultimate formation of stones in the stomach. Sarcinæ ventriculi have also accumulated in large numbers and formed a tumor.

An interesting case of the ingestion of foreign bodies by a *lunatic* is reported by Vandivert and Mills[3] from State Hospital No. 2, St. Joseph, Missouri. This patient died apparently of nephritis and there *was no suspicion of gastric trouble* until autopsy. The mass of foreign material lay on the sacculated portion, leaving a narrow channel along the lesser curvature for the passage of food. There was an atrophy of the mucous membrane, much formation of connective tissue, erosions, and the points of some of the foreign bodies had penetrated the stomach walls, but adhering omentum prevented leakage. There were some small walled-in abscesses. The appetite remained good until two weeks before death, and no symptoms pointed to the stomach. In all, 1446 objects were found in the stomach. As a matter of interest the list is given, also a photograph (Fig. 171), of the material found in the organ.

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[1] Med. Rec., Nov. 24, 1900.

[2] Centralb. f. Chir., Sept. 3, 1898.

[3] Jour. Amer. Med. Assoc., Jan. 21, 1911.

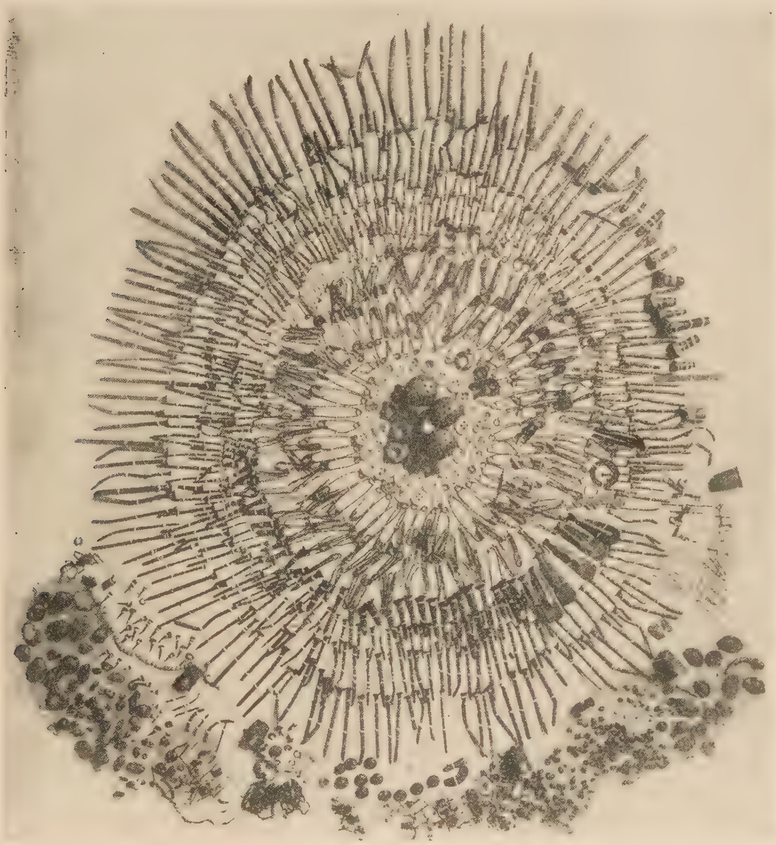

Fig. 171.—A human hardware store. A collection of foreign bodies found at necropsy in the stomach of an insane patient (Vandivert and Mills).

LIST

*Nails*

| | | |
|---|---|---|
| 20-penny | 5 | |
| 16-penny | 21 | |
| 10-penny | 24 | |
| 8-penny | 80 | |
| 5-penny | 113 | |
| 3-penny | 210 | |
| Total | 453 | 453 |

*Screws*

| | | |
|---|---|---|
| $2\frac{1}{2}$ inches | 4 | |
| $1\frac{1}{2}$ inches | 11 | |
| $1\frac{1}{4}$ inches | 5 | |
| 1 inch | 9 | |
| $\frac{3}{4}$ inch | 6 | |
| $\frac{1}{2}$ inch | 7 | |
| Total | 42 | 42 |

*Bolts*

| | | |
|---|---|---|
| $\frac{3}{8}$ x 1 inch | 3 | |
| $\frac{3}{8}$ x $1\frac{3}{4}$ inches | 1 | |
| $\frac{5}{16}$ x $2\frac{1}{2}$ inches | 1 | |
| $\frac{1}{4}$ x 2 inches | 1 | |
| $\frac{5}{16}$ x 1 inch | 3 | |
| Total | 9 | 9 |

*Miscellaneous*

| | | |
|---|---|---|
| Teaspoon handles | 5 | |
| Nail-file, $3\frac{1}{2}$ inches | 1 | |
| Pieces of steel, $2\frac{3}{4}$ inches | 3 | |
| Thimbles | 5 | |
| Salt-shaker tops | 3 | |
| Taps for $\frac{3}{8}$-inch bolts | 10 | |
| Buttons | 63 | |
| Safety-pins | 105 | |
| Hairpins | 115 | |
| Carpet-tacks | 52 | |
| Common pins | 136 | |
| Large white-headed pins, $2\frac{1}{3} \times \frac{1}{2}$ inches | 16 | |
| Needles | 37 | |
| Broken coat-rack hooks, 2 x $\frac{1}{4}$ inch | 7 | |
| String beads, small, 4 feet long | 1 | |
| Larger beads, loose | 70 | |
| Small stones and pieces of glass | 85 | |
| Prune seeds | 7 | |
| Pieces of metal, combined weight 3 ounces | 54 | |
| Hooks and eyes | 19 | |
| Grape, and other small seeds | 148 | |
| Total | 942 | 942 |
| Making a combined total of | | 1446 |
| Weight | 21,268 gm. | |

There may be local disturbances of severe type and vomiting, or if damage be done to the mucous membrane, then hemorrhage. On the other hand, there may be no disturbances at all, and the foreign body, especially if of small size and smooth, may be evacuated from the bowels. The history of the case will generally give us information. If the tumor be of sufficient size and very movable, it can be at times appreciated by palpation.

The $x$-rays will give information as to its presence.

**Treatment.**—The use of an emetic is objectionable, as a rule, unless the foregin body is extremely small and smooth. Personally, I never employ it. Mayou has recommended the use of an electro-magnet inserted in a stomach-tube, there being sufficient space at the end of the tube to draw in a small metallic object which can be located by the $x$-rays.

Under usual conditions, the administration of constipating food, potatoes, rice, and the soft part of bread, and keeping the bowels costive for several days, so as to form a protective mass about the foreign body, is the best method of treatment. It is an error to immediately administer a cathartic, as damage is done to the intestinal canal if there be any sharp edges to the object. Complications, such as perforation or inflammatory adhesions, are liable to occur if it be of any size. Intestinal obstruction may even result. If the body is of large size or serious symptoms ensue, early gastrotomy is indicated. Occasionally a small foreign body may be located by the gastroscope and removed through it by long forceps.

## CHAPTER XIV

# FUNCTIONAL DISEASES OF THE STOMACH

Under functional diseases of the stomach we may classify those affections in which either the secretory or motor functions of the stomach are at fault. Anatomic lesions are present in some cases and are absent in others.

The principal symptoms are due to the disorders of secretion or motility. Among these we classify hyperacidity (hyperchlorhydria), hypersecretion (gastrosuccorrhea), atony of the stomach, dilatation of the stomach, and achylia gastrica.

Many cases of hyperacidity and of hypersecretion are pure secretory neuroses; atony may also result from nervous disorders, but there are other causes for these conditions.

When there is dilatation due to stenosis of the pylorus, as from malignant tumor, the symptoms are due to the cancer and also to the relative motor insufficiency. It is difficult, therefore, to place any special disease of the stomach under a pure classification, owing to the diverse etiology. With achylia gastrica we have the loss of secretory functions of the stomach, and it may be classified under functional diseases. It is produced either by nervous influences or by anatomic changes in the gastric mucous membrane. As in many cases organic changes are present, I judged it advisable to devote to it a separate chapter.

### HYPERACIDITY (HYPERCHLORHYDRIA)

(*Synonyms.*—Hyperaciditas Hydrochlorica; Superacidity.)

The term "hyperchlorhydria" should be used to designate an increased secretion of gastric juice or, more correctly, of hydrochloric acid, during the period of digestion; that is, an overproduction of this acid. Under normal conditions the free hydrochloric acid fluctuates in the stomach within certain limits, thus:

Free HCl averages between 25 + and 50 +, or about 0.1 to 0.2 per cent., and the total acidity is from 40 + to 65 +, or 0.146 to 0.237 per cent.

We speak of hyperchlorhydria when not only the total acidity is higher than normal, but when the excess of free HCl is above normal. It is not sufficient to merely test for the total acidity, as it may be increased by organic acids. The free HCl should be determined quantitatively.

We might place a total acidity of 70+, of which the greater part consists of free hydrochloric acid, on the dividing line. A patient with such a degree of acidity and complaining of clinical symptoms may be considered a case of hyperchlorhydria.

A total acidity of 100 to 120, with free HCl, 80+ to 90+, is quite common, and much greater degrees of hyperchlorhydria have been observed.

We must remember that individual idiosyncrasies exist as to the susceptibility to free HCl. We occasionally find patients with a total acidity of 100+, the greater part of which is free HCl, who have no subjective disturbances whatever and never suffer any discomfort. In this regard an unsuual case is reported by Verbrycke,[1] a patient of Morgan, with free hydrochloric acid 154+, or 0.56 per cent., and a total acidity of 204+, or 0.74 per cent., with only slight pains after meals. One must also remember that there may be a small amount of gastric juice with high acidity, or a larger amount with lower percentage, but high total content of hydrochloric acid. Moreover, bacteria may be present when there is considerable hydrochloric acid, their presence then indicating diminished motility.

On the other hand, I have at present a patient under treatment with a total acidity of 60+ and free HCl 40+, who has all the symptoms of hyperchlorhydria.

**Frequency.**—It was formerly thought that in most diseases of the stomach the gastric secretion was deficient, but it has been demonstrated that the gastric juice is increased in about one-half the cases.

One does not see so many cases of hyperchlorhydria in hospital practice, except in connection with gastric ulcer, since the symptoms rarely become so severe as to require hospital treatment. As a concomitant of chlorosis it is frequently found. The gastric analysis in these cases is often neglected. Moreover, the general practitioner rarely examines the gastric contents in cases which present the symptoms of hyperacidity. It is, therefore, difficult to secure statistics as to its frequency.

Einhorn reports a trifle more than one-half his patients to be sufferers from hyperchlorhydria. Records of my private patients show that about 50 per cent. come to me for treatment for this condition.

**Etiology.**—No age is exempt. It is met with in adults and quite frequently in young people. In some cases it is a *neurosis*. Nervous excitement, violent emotions, sorrow, worry, and severe mental labor may be direct causes.

Neurasthenics and melancholics often suffer from it. It is a frequent concomitant of gastroptosis. In this condition the hyperchlorhydria is not the result of the nervous condition alone, but in part due to the ptosis, with resulting circulatory changes. Its

[1] N. Y. Med. Jour., July 16, 1910.

relief by Rose's belt is the proof, as reported in a series of cases at the Manhattan State Hospital.[1] It occurs with mucous colic, in which case I believe gastroptosis is a factor.

Chlorosis is a frequent cause. Irritation of the mucous membrane of the stomach may be a direct factor, as from bolting the food, large quantities of cold or hot drinks, alcoholic excess, pickles, rich condiments, and insufficient mastication.

Special articles of food or drink are often productive of it, such as very strong coffee. Excessive smoking and chewing of tobacco are also factors.

Cholelithiasis and nephrolithiasis are said to be causes.

Hyperchlorhydria is a frequent accompaniment of gastric ulcer. This is particularly true of the acute ulcer. It may accompany duodenal ulcer, and is found with benign stenosis and dilatation of the stomach, and with gastrosuccorrhea. It is more frequent among the wealthy class, such as bankers, brokers, and professional men, though it occurs among the poor. There seems to be no special predilection for either sex.

Hyperchlorhydria may also occur as a reflex from appendicitis or gall-bladder disease. Spasm of the pylorus may accompany these conditions.

**Symptoms.**—They rarely appear suddenly, but usually develop gradually. They always come on after eating and never on an empty stomach, after the time for digestion has elapsed. At first the patient complains of a disagreeable sensation or an uneasy feeling about one to three hours after a meal. It may be a feeling of pressure or fulness, or of heat, or of tingling. This increases into a feeling of distress in the epigastric region, or a burning sensation after each meal. In the mild cases there is no actual pain.

In severe cases pain may be marked, with acid belching and heart-burn, and the patients suffer severely and in some cases appear almost in a state of collapse. The burning may be felt in the esophgus or along the back and is due to the eructation of the acid contents. Violent headache often accompanies it. Some claim they can feel spasmodic movements in the stomach. This is due to contraction (spasm) of the *pylorus* and an increased peristaltic action of the organ endeavoring to overcome the obstacle. Mild attacks of pain last for a brief period of time, while the more severe attacks persist for several hours.

Vomiting occurs occasionally during the height of the cardialgic attacks. The vomitus is very acid and burning, and after its occurrence relief, as a rule, results.

In some cases the pain appears after eating certain articles of food, and patients seem to have idiosyncrasies, such as to coffee, etc. The attacks are not always directly dependent on the degree of the digestibility of the food, and at times they can eat indigestible material without discomfort; whereas at other times digestible articles of diet will cause pain. Probably the nervous element plays a part in these cases, causing undue irritation of the nerves of secretion.

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[1] Rose and Kemp, Atonia Gastrica.

Some suffer more disturbance after a small meal, while after a large meal they have no distress. This is readily explained by the fact that the larger amount of food combines with a greater portion of the free HCl secreted.

Patients when the symptoms first appear can ease their pain by taking nourishment, especially if rich in albumin, as the whites of eggs and milk or meat. It also disappears after the administration of an alkali, such as milk of magnesia (Phillips), Vichy, or soda bicarbonate. Persons living chiefly on a starch diet suffer more intense pain than those who live largely on meat and eggs; hence, the character of the food has frequently a relation to the pain.

*Appetite.*—In most cases the appetite is very good, and in some is greatly increased. Others are readily satiated, but the desire for food soon comes on again.

*Thirst* is at times increased during the active attack, but in many cases is normal. If hyperchlorhydria is complicated by dilatation of the stomach, thirst is present.

*Bowels.*—Constipation is, as a rule, present, though constipation and diarrhea may alternate.

*Headache.*—Severe headache often occurs, or even attacks of dizziness, generally during the occurrence of gastric pain.

*Nutrition.*—These patients generally do not lose in weight nor present the aspect of being very sick. They are rarely particularly well nourished and are often of nervous temperament or anemic. This is not invariable, as one has at times to treat stout, well-nourished men, high livers, and inveterate smokers and drinkers, who suffer from this complaint. Rarely, under improper diet, loss of weight may occur.

*Nervous Symptoms.*—Some patients are depressed, nervous, suffer from insomnia, and are neurasthenic. Migraine occurs in others.

*Urine.*—Acidity of the urine may be reduced during the course of digestion. There are no characteristic features.

*Physical Examination of the Stomach.*—During intervals between attacks there may be nothing in evidence.

*Percussion.*—At the time of the attack the stomach may be distended.

*Palpation.*—The greater part of the gastric region will be found to be tender on pressure or even painful in some cases. The tenderness covers a large area, generally over the greater part of the stomach; often in the region of the pylorus this tenderness is accentuated.

*Splashing Sound.*—This can be produced after meals or after the ingestion of water, but is not present in the fasting condition.

*Examination of the Stomach-contents.*—This is the decisive test. If the stomach is aspirated in the fasting condition, it is found to be empty, or only a few cubic centimeters of juice can be secured.

One hour after Ewald's test-breakfast or three to four hours after the Leube-Riegel test-dinner the stomach-contents show an extremely acid reaction, often two to three times higher than normal (from 100+ to 150+).

It is not sufficient to determine the total acidity, but the value of the free hydrochloric acid is the important feature. Only in hyperchlorhydria is it increased, and in this condition the free HCl causes the high degree of acidity; and Töpfer's test (dimethyl-amido-azobenzol) shows that the free hydrochloric acid constitutes the chief content, and is often within 10+ to 15+ of the total acidity. Mintz's method is also excellent.

The digestive power is very good; a small disk of egg-albumen will be digested in a short time, sometimes within one-half to one hour. Three to four hours after the test-dinner meat will be found to be perfectly digested, while starchy substances will be at times unchanged or little altered.

Starch or erythrodextrin will be found, Lugol's solution giving a blue or dark red reaction. If, however, a small amount of starchy food be given with a large amount of albuminous material, while the HCl is entering into combination and before free hydrochloric acid appears, the normal reduction changes in the starch may occur. With large quantities of amylaceous material the brief period before the appearance of hyperchlorhydria will not allow a complete conversion of all the starch, and this tends to remain in the stomach an abnormally long time and to produce fermentation.

A. L. Benedict[1] describes an effervescent test for gastric acidity. He administers 30 to 50 c.c. of a strongly saturated solution of sodium bicarbonate, to be taken at a single gulp, and, preferably, by means of a stethoscope, determines, by the degree of effervescence, the degree of acidity. This method is only relative. Boardman Reed has experimented with the method. The writer does not, however, recommend it.

*Absorption* from the stomach is not disturbed; potassium iodid appears, if anything, more rapidly in the saliva.

*Motor Function.*—This is not impaired, but is often rather increased. Two hours after the test-breakfast or six to seven hours after the test-dinner the organ is found to be empty or nearly so. Salol appears as salicyluric acid in the urine an hour after its ingestion when tested for with ferric chlorid solution. We must remember that hyperchlorhydria untreated, with fermentation resulting from improper diet (excessive starch), may in some cases produce atony and resulting insufficiency. Hypersecretion, which may accompany hyperchlorhydria, is another factor.

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[1] N. Y. Med. Jour., May 11, 1911.

**Course.**—It may be rapid, coming on suddenly and lasting a short time, or chronic, for months to years. It often varies in the early stages and may be intermittent in its character. The attack may last for days, weeks, or even months, and then there be a free interval for a considerable period of time. It can recur without any apparent cause, or result from some mental shock or worry, or from some dietary indiscretion. Gradually the hyperchlorhydria becomes more frequent and at last the condition becomes permanent.

In rare cases the attacks appear to come on later than usual, and vomiting of an acid mass of undigested food may occur, showing the contents were retained an abnormally long time. This is undoubtedly due to spasmodic closure of the pylorus from irritation by the acid contents.

Atony and even atonic dilatation may develop from this type, and hypersecretion (gastrosuccorrhea) may result in some cases.

**Prognosis.**—The prognosis is, as a rule, good. Many patients can be completely cured. In some very old or severe cases there is a tendency to relapses. The disease is not dangerous to life.

Those in whom the nervous element plays a part often do not readily respond to treatment and are a source of discouragement to the physician. Relapses, in spite of the greatest care and for no apparent reason, are not infrequent.

If atony or dilatation of the stomach complicate the hyperchlorhydria, the prognosis as to cure is not as favorable.

**Pathology.**—As the cases are not fatal, it is hence unknown. In one case, dying of intercurrent disease, a few erosions were found. It is evidently a disorder of the secretory function.

**Diagnosis.**—The diagnosis of hyperchlorhydria can be made from the examination of the gastric contents:

1. An hour after Ewald's test-breakfast the acidity is found increased, due to the increased quantity of free hydrochloric acid.

2. In the fasting condition the stomach is found empty or nearly so. (This excludes gastrosuccorrhea.)

Furthermore, the patient suffers from certain subjective symptoms:

1. Discomfort or, *more generally, a pain* which comes on from one to three hours after a meal. This is directly dependent upon the ingestion of food. It is not a continuous pain and there are intervals of relief. The character of the food and the quantity usually have a bearing. Starchy food readily produces it, and a light meal more than a full meal. The latter, when rich in albumin, often causes no disturbance. The pain may be " a dead, dull pain, with a gnawing sensation," or may be of a burning character. The administration of an alkali, such as Vichy or sodium bicarbonate, relieves the pain by neutralizing the acid; or milk, raw eggs, or a meat sandwich, by binding the free acid, produces the same result.

2. In others there is more the feeling of a heat or burning in the stomach or the feeling of a sour stomach.
3. Appetite and thirst are generally normal.
4. No cachexia.
5. Marked constipation is the rule.
6. Some cases are quite nervous or even neurasthenic.

**Complications.**—It is often necessary to determine whether atony, dilatation of the stomach, or gastroptosis are associated with the hyperchlorhydria.

*Simple Atony.*—Presence of the splashing sound and 100 c.c. or more of gastric contents aspirated one hour or more after Ewald's test-breakfast; or the splash with the presence of food in the stomach six or seven hours after the test-meal; the lower border is in normal position as determined by the splash and by percussion.

*Dilatation.*—Splash at the umbilicus or below shows descent of lower border. Percussion alone or with inflation, or gastrodiaphany substantiate this finding; there is no descent of the upper border. Kidneys are normal in position.

*Gastroptosis.*—Splash at the umbilicus or below it shows descent of lower border.

*Movable kidney is diagnostic if found in addition.* There is diastasis of the recti muscles; inflation or gastrodiaphany shows descent of the upper border of the stomach, but are scarcely necessary. The *x*-rays are unnecessary for diagnosis.

**Differential Diagnosis.**—*Acid Gastritis.*—Hyperacidity is present plus abundant stomach mucus.

*Hypersecretion* (*Gastrosuccorrhea*).—The stomach in the fasting condition contains 75 to 100 c.c. or more of very acid gastric juice. The persistent appearance of 20 c.c. of gastric juice in the fasting stomach is considered pathognomonic by many. Severe attacks of pain and vomiting generally occur during the night or early morning.

*Biliary Colic.*—Pains are later (four to five hours after meals) or independent of meals. The pains of hyperchlorhydria are dependent on the meals. Pains of biliary colic are not relieved by alkalis, and they extend over the right epigastrium and hypochondrium, and frequently to the right shoulder or right axillary region. A patient with biliary colic or even after the attack has no appetite and cannot eat.

The appetite is good in hyperchlorhydria.

The gall-bladder is painful on pressure and at times swollen. Icterus may be present. Gall-stones at times are found in the stool. Leukocytosis is present in biliary colic if inflammation is associated. Sometimes the differential diagnosis is difficult. Gastric analysis may be necessary to determine it, though occasionally both conditions occur together. The presence of Head's gall-bladder zone may aid diagnosis.

*Ulcer of the Stomach.*—Epigastric pain is intense, appears shortly after the ingestion of food; local tenderness on pressure and pain increased thereby. Pain disappears at the end of digestion. There are the dorsal pain and vomiting in many cases soon after meals.

Hematemesis occurs or occult blood is found in the gastric contents or stool. Occult blood may occur with gall-bladder disease in the gastric contents of achlorhydria hæmorrhagica gastrica, a disease of peculiar characteristics already described, and not particularly difficult of diagnosis. Ulcer is more frequent in women. The *discovery of occult blood* is often the *determining factor* in our diagnosis.[1]

*Nervous Gastralgia.*—More frequent in women from eighteen to thirty years. Pain appears without regularity, and is in no way dependent upon the meals or character of food. Is relieved by pressure. Intervals of perfect health; nervous temperament always present. Gastric analyses often vary in the same case.

**Treatment.**—The treatment comprises, first, the removal of the causes of hyperchlorhydria; and second, the cure of the condition itself.

*Removal of the Causes.*—Interdict tobacco smoking and chewing. If a cigar or cigarette holder is employed, smoking once or twice a day I believe may be harmless, as it prevents swallowing saliva impregnated with tobacco juice, the chief source of irritation in my opinion. Alcohol in every form, including wines and beers, should be prohibited. All kinds of acids, such as acetic, tartaric, or citric, should be forbidden; and all foods prepared with them, such as with vinegar or lemon-juice; and all acid or acidulated drinks.

Condiments, such as pepper, ginger, horseradish, etc., pickles, mustard, paprika, nuts, acid fruits, grapefruit, and radishes, should be prohibited.

Avoid all extremes of heat and cold in food and drink.

Thorough mastication of the food should be enjoined.

Nervous conditions, when present, should be treated.

Hygienic regulations are important.

Overwork and mental anxiety are factors in the production of hyperchlorhydria. Brokers, professional, and business men must be relieved temporarily from overwork and worry by being sent into the country, where various open-air amusements, such as golf, horseback riding, driving, walking, etc., can be indulged in. If the patient have a taste for fishing or shooting, such diversions are excellent. I have seen a few weeks in camp work wonders.

Those indulging in a continuous round of social festivities should be compelled to lead a quiet life.

On the other hand, there are many people of wealth with no occupation whatever who become entirely self-centered and nervous therefrom, and for such a class of persons occupation is of great value.

Hydrotherapy, such as sponge-baths, douches, etc.; also a moderate indulgence in calisthenics (ten minutes morning and night).

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[1] The presence of pus in the gastric contents aids diagnosis.

For the cure of the hyperchlorhydria there are practically two methods used, alone or in combination:

We may bind the excess of free hydrochloric acid by the administration of large quantities of proteins or we may neutralize the excessive acid by the administration of an alkali. Clinical observation has demonstrated that those articles of food which are capable of binding large quantities of HCl are borne the best. The burning feeling of distress or pain is *relieved by the administration of albuminous food.* Carbohydrates, if given in any quantity, cause distress. The diet is, therefore, of greatest importance.

*Diet in Hyperchlorhydria.*—As noted under Prophylaxis, all articles which are liable to overstimulate the secretory glands of the stomach should be forbidden. Among such are acids, all spices, as pepper and mustard, pickles, horseradish, olives, acid fruits, beers, and wines.

The food should be rich in albumin, such as chops, steak, roast beef, mutton, game, eggs, milk, oysters, and fish. Bread and butter can be taken, the former in moderation. Green vegetables, such as spinach, asparagus, lettuce, peas and string beans, potatoes, rice, and other cereals, should be given in small quantity. They should be taken in combination with large amounts of albuminous food.

*Alcohol in all forms,* including beers and wines, I believe should be avoided, also coffee; though some allow a small amount of beer and very weak coffee. Fieiner has demonstrated that egg-albumen binds more free hydrochloric acid than any other food. Among other articles especially suitable for this purpose he recommends boiled veal, beef, mutton, raw ham, Leube-Rosenthal's beef solution, boiled ham, boiled pork, Swiss cheese, Roquefort, pumpernickel, milk, and cocoa.

In my own experience I have found gelatin[1] an excellent remedy, employing 1 or 2, or even 3 ounces of a 5 to 10 per cent. gelatin solution, flavored with a pinch of sugar or a little vanilla, and given midway between meals. The value of egg-albumen and cocoa is marked. Starchy foods that have been well dextrinized, such as zwieback and toast, subsequently dried in the oven, are more readily digested.

Strauss has shown that if carbohydrates are introduced in the form of sugars in solution they do not markedly increase HCl secretion. He gives 200 to 300 c.c. of a 20 per cent. dextrose solution during the day.

Considerable water should be taken during meals, or Appollinaris and seltzer, if no atony is present.

Fats, such as butter and cream, are of value. Since the carbohydrates are restricted and additional calories must be secured, Strauss, Ewald, and others advise their use. Furthermore, fats lessen the acidity, also possibly the irritable tendency of the mucous membrane. Cream has been thus recommended.

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[1] Calves' foot and chicken jellies are excellent.

For some years I have been accustomed to administer olive oil once or even three times daily before meals in obstinate cases of hyperchlorhydria, using from ½ to 1 ounce (16.0–32.0), suspended in water. This has lessened the hyperacidity. In the same way, glycerin, ½ to 2 drams (2.0–8.0), mixed in water, may be used. Illoway[1] employed it in one case. Almond oil is also of value.

If we attempt to treat hyperchlorhydria by diet alone, we should give three additional feedings at a time after the regular three meals, such as would bind the excessive hydrochloric acid and prevent the symptoms. The extra feedings may consist of koumiss, matzoon and Vichy, bacillac, bouillon, a sandwich, milk, raw eggs (especially the whites) and milk, with crackers or bread and butter. One can select a diet from the tables with a sufficient number of calories.

For practical purposes an improvement in nutrition—*i. e., some increase in weight*—should be secured, even though slight, in addition to the amelioration of symptoms. This refers only to those of thin habit, not to the well nourished. The use of the scales, weighing at stated intervals, is of radical importance. Assimilation differs in individual cases, and though on paper the calories may be correct, a *loss of weight* shows a radical error in the treatment. The following is a sample dietary, such as is usually recommended for a patient of good physique and quite active. The content of protein is very high as compared with Chittenden's scale, which I advocate in health. It is frequently advisable to diminish the quantity of meat and substitute milk, matzoon, koumiss, or bacillac. Latcone-buttermilk is also excellent. Gelatin solutions bind the free hydrochloric acid in a satisfactory manner. Depending on the normal weight, height, and occupation, the diet must be formulated in every case:

| | | Calories. |
|---|---|---|
| 7.30 A. M.— | 250 c.c. milk, cocoa, 2 eggs, 3 zwieback, and butter (20 grams) | 690 |
| 10.30 A. M.— | 200 c.c. milk, with 1 raw egg, or matzoon and Vichy 125 c.c., or koumiss 250 c.c., or milk 250 c.c., or bouillon with 1 raw egg (approximately) | 240 |
| | Bread 2 slices, or crackers 2 oz. (gm. 60) | 160 |
| | Butter gm. 20, water, or Appollinaris, or Vichy 250 c.c., occasionally weak tea | 163 |
| 1.30 P. M.— | Chops, steak, beef, or mutton, 100 gm. (about) | 200 |
| | Mashed potatoes, 30 gm. | 37 |
| | Spinach, 30 gm. | 12 |
| | Bread (1 slice), 30 gm. | 81 |
| | Butter, 10 gm. | 80 |
| 4.30 P. M.— | Same as at 10.30 A. M. | 563 |
| 7.00 P. M.— | Soup (barley), 200 c.c. | 100 |
| | Meat, broiled, 100 gm. | 200 |
| | Spinach or peas, 50 gm. | 30 |
| | Potatoes mashed with milk, 50 gm. | 80 |
| | Weak tea, 100 c.c. (three-fourths milk) | 64 |
| | Toast (1 slice) | 75 |
| | Butter, 10 gm. | 81 |
| 10.00 P. M.— | Milk and Vichy, āā 100 c.c. | 64 |
| | Total calories | 2920 |

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[1] N. Y. Med. Jour., May 25, June 1, 15, and 29, 1901.

Various modifications may be made.

Coffee I interdict. All fried food should be forbidden, as should hot breads, hot biscuits, and rich desserts. The food should not be excessively hot or cold. The patient should eat slowly, masticate thoroughly, and rest after eating for at least twenty minutes to one-half hour.

Illoway and Bouveret advocate only three meals a day, with a sandwich at bedtime in some cases. They believe the stomach should have an interval of rest so as to become perfectly emptied. Practically, Illoway's only medication is one-half glass of French Vichy, given at 10 to 11 A. M., at 4 to 5 P. M., and at night, if the sandwich is omitted.

They believe that unless the stomach have some rest, atony or atonic dilatation may result.

On the other hand, some patients can only eat a small amount of food at a time, have the desire to eat at frequent intervals, and feel better if they do so. Repeated feedings are difficult to follow out in some cases, so conditions vary.

My method is to give the three regular meals a day with diminished carbohydrates, with the alkalis an hour to an hour and a half or even two hours later. If it is necessary to improve nutrition, additional feedings are given, a glass of koumiss, or milk and raw egg, or matzoon and Vichy, or 1 to 2 ounces of 10 per cent. gelatin solution (sweetened) between meals, or if pain comes on after the administration of the alkali. These foods can be alternated.

*Medication.*—Among the alkalis that are of value are Vichy, imported or siphon, bicarbonate of soda, milk of magnesia (Phillips), magnesia usta, magnesia ammoniophosphorica, and biborate of soda (Jaworski).

A little sugar of milk can be added to the soda or magnesia preparations to make them more agreeable. If the hyperchorhydria is complicated by atony, then bicarbonate of soda (which generates considerable carbonic acid gas) would be objectionable.

The following rule should be carried out as regards the administration of the alkalis: "Give them in ample time to anticipate the appearance of symptoms by a considerable margin." They should be administered t. i. d. after meals, depending upon the time of appearance of symptoms. Repeat the dose if the symptoms begin later, unless albuminous food is given as a substitute.

The magnesia preparations have the tendency to move the bowels and hence are of special value. If the result is too active, combine the magnesia with varying proportions of soda bicarbonate, or give the latter alone, or give bismuth subnitrate with the magnesia. Magnesia usta will neutralize about four times more acid than soda bicarbonate.

The dosages are as follows, given from one to two hours after meals t. i. d. :

| | |
|---|---|
| Vichy | 175 to 250 c.c. (½–1 glassful). |
| Vichy | ½ to 1 glassful plus sod. bicarb. ½ to 1 teaspoonful (2.0–4.0). Allow this to become flat (effervescence to disappear). |
| Sod. bicarb. | ½ to 1 (2.0–4.0) teaspoonful in one-third glassful of water. |

| | | |
|---|---|---|
| ℞. | Magnesia usta<br>Sod. bicar. | āā |

Dose, ½ to 1 teaspoonful (2.0–4.0) in water t. i. d.

A little sugar of milk or 1 minim of oil of peppermint may be added to flavor.

If the bowels move too freely, diminish magnesia, then give:

| | | |
|---|---|---|
| ℞. | Magnesia usta | ℥ss (16.0); |
| | Sod. bicarb. q. s. | ℥ij (64.0).—M. |

Dose, ½ to 1 teaspoonful (2.0–4.0) in water.

If necessary, bismuth subnitrate, 2 drams to ½ ounce (8.0–16.0), may be added.

| | | |
|---|---|---|
| ℞. | Sod. bicarb. | ℥j (32.0); |
| | Magnesia usta | ℥ss (16.0); |
| | Magnesia ammon. phos. | ℥ss (16.0).—M. |

Dose, ½ to 1 (2.0–4.0) teaspoonful in water.

| | | |
|---|---|---|
| ℞. | Magnesia usta | ℥ss (16.0); |
| | Pulv. rhub. | gm. xv (1.0); |
| | Soda bicarb. | ℥ss (16.0); |
| | Sugar of milk | ℥ss (16.0).—M. |

Dose, ½ teaspoonful (2.0) in water if costive, or,

| | |
|---|---|
| Milk of magnesia (Phillips) | ʒj to iv (4.0–16.0), in water t. i. d. |

This last I have found to be an excellent preparation, especially where constipation is marked. It is one of the most serviceable remedies for hyperchlorhydria.

| | |
|---|---|
| Sod. biborate | gr. x to xv (0.6–1.0), in water (Jaworski), |

has been recommended.

The use of Carlsbad water or a glass of the imported Carlsbad salts, 1 to 2 drams (4.0–8.0) in warm water on rising, lessens acidity and helps the bowels.

Wolff's artificial Carlsbad mixture consists of:

| | | |
|---|---|---|
| ℞. | Sod. sulph. | 30.0; |
| | Sulph. potass. | 5.0; |
| | Sod. chlorid | 30.0; |
| | Sod. carb. | 75.0; |
| | Sod. biborate | 10.0.—M. |

He adds sod. biborate on account of Jaworski's recommendation.

Dose, ½ dram (2.0) in one-half glassful of warm water two hours before meals.

In some cases I give olive oil, ½ to 1 ounce (15.0–30.0) or more, or glycerin, ½ to 2 drams (2.0–8.0), in a little water before meals, to lessen gastric irritability; bismuth subnitrate, 15 grains to $\frac{1}{2}$ dram (1.0–2.0), given with the olive oil, is often useful.

If the pain is severe, the administration of belladonna before secretion commences, in order to lessen the gastric juice, is of value. At the time of pain it is too late. It is serviceable also in obstinate cases, thus before meals:

℞. Tinct. belladonna........................ ʒiss (6.0);
Aq. destil..........................q. s. ℥ij (60.0).—M.

Dose, $\frac{1}{2}$ to 1 teaspoonful (2.0–4.0), or larger dose of tinct. belladonna, up to 10 drops, may be given.

or

Ext. belladonna.......................... gr. $\frac{1}{6}$ to $\frac{1}{3}$ (0.01–0.02).

or

Atropin.............................. gr. $\frac{1}{100}$ (0.0006).

Illoway suggests:

℞. Tinct. aconite root....................... 6 drops;
Tinct. belladonna........................ 25 drops;
Aq. destil..........................q. s. ℥j (30.0).—M.

Dose, 1 teaspoonful (4.0) on rising and a second dose in half an hour. No more.

If the pain is extremely severe and not relieved by an alkali or belladonna, a small dose of codein, $\frac{1}{8}$ to $\frac{1}{4}$ grain (0.008–0.016), may be required. This may even be given by hypodermic with excellent results. Morphin in same dosage is rarely necessary and should only be given by the physician.

The application of heat to the epigastrium by moist compresses, hot-water bag, or poultice is of service.

If the patient is very restless and disturbed, one of the bromids—strontium bromid, sodium bromid, or ammonium bromid—may be given in 10- to 15-grain (0.06–1.0) doses for a brief period.

In obstinate cases, silver natrate, $\frac{1}{8}$ to $\frac{1}{4}$ grain (0.008–0.016) t. i. d., or in solution, has been recommended to relieve gastric secretion, or occasionally a douche or spray with silver nitrate (1 : 2000), followed by lavage with water. If the douche is used, lavage is unnecessary unless there is a complicating atonic dilatation. If one sees the patient during a severe attack of pain, aromatic spirits of ammonia, $\frac{1}{2}$ to 1 dram (2.0–4.0), diluted well with water; lime-water, 1 to 2 ounces (30.0–60.0), mistura cretæ, $\frac{1}{2}$ to 1 ounce (15.0–30.0), or bicarbonate of soda or the magnesia preparations can be given at once.

Sometimes sodium bicarbonate distends the stomach and causes more pain. Emesis is often a relief, and at times, when other treatment fails, lavage may be used as a temporary method.

*Electricity* has been recommended, especially by Einhorn, for the treatment of hyperacidity.

Internal galvanization has been suggested for the relief of pain. It seems to be impractical.

In cases when constipation is obstinate or when atony is present, it might be of service, either the intragastric or, preferably, the percutaneous method of faradization.

*Bowels.*—Massage, vibratory massage, and rectal injections of olive oil are of service, as well as out-of-door exercise, horseback riding, etc. Remedies such as cascara, aloes, phenolax, purgen, regulin, etc., may be at times temporarily necessary.

If there are atony, dilatation, or ulcer, these conditions must be treated. Rose's belt is indicated for atony or atonic ectasia.

To briefly recapitulate. I have found the following of value:

Carlsbad salts on rising.

Milk of magnesia (Phillips) or magnesia usta, alone or with soda bicarbonate, or Vichy and soda bicarbonate, one to two hours after meals; midway between the chief meals a little milk and raw eggs, koumiss, matzoon, and Vichy, or 1 to 2 ounces (30.0–60.0) of gelatin solution (5 to 10 per cent.).

In some cases olive oil before luncheon and dinner, and belladonna extract or tincture when required.

The proper diet must be followed.

## GASTROSUCCORRHEA (CONTINUOUS SECRETION OF GASTRIC JUICE)

(*Synonyms.*—Hypersecretion; Parasecretion; Excessive Flow of Gastric Juice; Reichmann's Disease; Gastroxynsis—Rossbach.)

Gastrosuccorrhea is a perversion of function in which the glands of the stomach secrete large quantities of gastric juice even when the *stomach is empty*, and hence when there is no irritation from ingested food. The diagnosis rests on the removal from the stomach in the fasting condition of a considerable quantity of gastric juice, with the addition of symptoms which present a characteristic picture. To Reichmann the credit is due of having first called attention to this perversion of function.

Gastrosuccorrhea may be classified in two types:

1. Gastrosuccorrhea continua periodica or the intermittent form of hypersecretion; the attacks occur at irregular intervals.
2. Gastrosuccorrhea continua chronica or chronic hypersecretion.

Though some believe that hypersecretion is a purely secretory neurosis, other factors can also produce it. Unquestionably, nervous conditions, such as mental excitement or mental overexertion, may be the direct cause in some cases; hence, gastrosuccorrhea may be a pure neurosis.

On the other hand, direct irritation of the mucus membrane can be the cause; in fact, the same factors that produce hyperchlorhydria, such as rapid eating, indigestible food, spices and condiments, abuse of alcohol, bolting the food, excessively hot or cold food or drink, etc.

Hyperchlorhydria of long standing, especially if neglected, may be a factor in the ultimate production of continuous secretion. In some of the gastric crises of locomotor ataxia gastrosuccorrhea is at times observed.

Hypersecretion is also at times an accompaniment of dilatation of the stomach, either of the atonic type or, more frequently, in the form due to stenosis of the pylorus. It also may occur with ulcer of the stomach. It can even accompany acute dilatation of the stomach, especially in that form engrafted on a chronic stenotic dilatation, to which I refer under Acute Ectasy. Tetany may be rarely associated.

In many cases of hypersecretion disturbances of the motor functions of the stomach are present in addition.

## GASTROSUCCORRHEA (CONTINUA PERIODICA)

(*Synonyms.*—Intermittent Secretion of Gastric Juice; Intermittent Hypersecretion; Gastroxynsis; Periodic Continuous Flow of Gastric Juice.)

This type of hypersecretion is characterized by an acute attack of continuous secretion of gastric juice associated with severe pains in the stomach, usually spasmodic in character, and by vomiting of acid fluid, the attacks generally occurring in the night or early morning and at irregular intervals; hence it is known as intermittent or periodic. Probably this condition is more frequent among nervous cases than we generally suppose. I have been able to absolutely determine in several cases of supposed intermittent attacks of acute gastritis, or so-called acute bilious attacks, that this condition of intermittent hypersecretion was present.

**History.**—Reichmann[1] was the first to describe this perversion of function, and Rossbach,[2] under the nomenclature gastroxynsis, described what is generally considered the same disease. Sahli, Riegel, and many others have written on it.

**Etiology.**—In some cases it is a neurosis, or the result of mental overexcitement or of overexertion; irritation of the gastric mucous membrane, as from cold water, or smoking, spices, etc., may precipitate an attack.

It is associated with the gastric crises of locomotor ataxia in some patients, or with organic affections of the peripheral or central nervous system, with gastric ulcer, or with the stenotic form of ectasia or with acute dilatation of the stomach. These are not pure cases, but have the additional symptoms incident to the disease.

**Symptoms.**—These usually begin during the night. The patient, who is generally apparently perfectly well, suddenly begins to feel discomfort in the gastric region, which is rapidly followed by pain of severe type and generally spasmodic in character. There are nausea, a feeling of faintness, and the patient is obliged to assume the recumbent position. He grows pale, the extremities become cold, and the abdomen at times is sunken and the pulse rapid and feeble. The nausea becomes worse and worse and soon a violent attack of vomiting of a large amount of acid fluid takes place.

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[1] Berl. klin. Wochenschr., 1882, No. 40.

[2] Deutsch. Arch. f. klin. Med., 1883, Bd. 35.

There is temporary relief, but the symptoms begin again, and after a short period the patient again vomits up a large quantity of fluid, far out of proportion to the amount previously ingested.

During the attack the appetite is lost and there is extreme thirst. Severe headache and constipation generally accompany these attacks.

These attacks generally occur in the middle of the night or early in the morning, and awaken the patient by the pain, if he be sleeping.

During the attack the stomach is tender on pressure and there is a good deal of heart-burn and acid belching, the urine is scanty, alkaline, and of high specific gravity.

*Character of the Vomitus.*—The fluid is watery and very acid. It may be clear or somewhat tinged with bile (yellowish green).

There may be particles of food in the first vomit if motor insufficiency is associated with the condition, but in many cases there is simply the clear gastric juice alone or mixed with bile. If this fluid be examined it will be found to contain free hydrochloric acid in considerable quantity, rennet, and pepsin. The desire to vomit frequently persists and generally several attacks of vomiting succeed each other. Even though the patient abstain from all fluid, in a few hours or less he will again vomit a large quantity of gastric juice. Rarely the vomitus may contain traces of blood, which does not necessarily mean an ulcer. This condition may last for several hours or for several days, when gradually the vomiting stops, the nausea and pain subside, and the patient begins to desire food. Gradually the appetite returns, the food is retained, in a few days he begins to feel nearly well, and in a brief time is apparently in perfect health.

The patient may continue in good health for weeks, months, or a year, and then have a recurrence. The intermissions of good health, on the other hand, may become shorter until, finally, the intermittent gastrosuccorrhea becomes chronic.

In many cases during the intermissions, if the gastric contents are analyzed, they will be found to be perfectly normal. On the other hand, some cases may suffer from mild symptoms of hyperchlorhydria, and such a condition will be found to exist on examination.

**Diagnosis.**—This can be made by the characteristic symptoms beginning during the night, the vomiting of pure gastric juice occurring when no ingesta are present in the stomach. Analysis shows that it possesses all the properties of the gastric juice with an excess of free hydrochloric acid. If no food is given and the stomach be aspirated before the second attack of vomiting occurs, at the time of appearance of pain and nausea, or if the second vomitus be analyzed, and in either event be found to consist of a considerable quantity of pure gastric juice, the diagnosis is conclusive.

The attacks are intermittent.

All cases should be examined as to motor functions and dilatation, since attacks of gastrosuccorrhea occur with these conditions. One should also exclude organic disease, such as the gastric crisis of locomotor ataxia. In the latter case we have loss of patellar reflexes, the Argyll-Robertson pupil, and the Rhomberg symptom.

**Prognosis.**—In pure cases of gastrosuccorrhea continua periodica the prognosis may be fairly good. It is often possible to lessen the severity of the attacks or in some cases even to effect a cure.

**Treatment.**—*Prophylaxis.*—We must first endeavor to find the cause of these attacks, and by correcting it prevent their occurrence. In the interval between attacks the stomach-contents should be examined after a test-breakfast or meal, and we should determine whether or not hyperchlorhydria exists.

The motor functions of the stomach should also be carefully tested, and any motor insufficiency if present should receive treatment. If there be excesses in smoking or drinking, tobacco and alcohol should be cut off. If there are errors in diet, they should be corrected. If mental overexertion or nervous excitement is a cause, such conditions should be corrected. If the patient is neurasthenic, he should receive careful treatment. Hygienic method of living, exercise out of doors, horseback riding, golf, etc., are serviceable.

*Treatment of the Attack.*—During the early stage, when the pain and nausea first begin, binding the free acid with the whites of several raw eggs beaten up in water or in milk, or the use of 1 to 2 ounces (30.0–60.0) of 10 per cent. gelatin, or neutralizing the acid by the administration of an alkali, such as ½ to 1 dram (2.0–4.0) sodium bicarbonate in water, 4 ounces (125.0), or Vichy; or milk of magnesia, ½ ounce (16.0), or magnesia usta, ½ to 1 dram (2.0–4.0), will at times mitigate the symptoms. Einhorn recommends a large dose of bromid at the appearance of the first symptoms, and claims that it will often cut the attack short or lessen its severity. About 15 to 30 grains (1.0–2.0) of sodium bromid should be given, preferably in Vichy (an alkali).

The patient should be kept recumbent, with the application of moist heat or dry heat (wet hot flannel compress or hot-water bag) applied to the gastric region. He will generally vomit in spite of treatment.

The best method, I believe, is to perform lavage *early*, not waiting until vomiting occurs, if the *pain and nausea are not* relieved by administration of the albuminous food or alkalis.

Wash the stomach thoroughly with an alkaline solution—1 to 2 quarts (liters) of warm water in which 2 to 3 ounces (30.0–45.0) of milk of magnesia (Phillips) have been dissolved—or ½ ounce (16.0) of sodium bicarbonate or ½ ounce (16.0) of magnesia usta. Before withdrawing the tube after lavage, pour through it into the stomach 2 drams (8.0) of milk of magnesia dissolved in 2 ounces (30.0) of water, to which is added 10 drops (0.6) tincture of belladonna to check further secretion. Sodium bicarbonate, 1 dram (4.0), may be substituted for the magnesia.

If the pain is very severe it may be necessary to give one or several hypodermics of morphin, $\frac{1}{8}$ to $\frac{1}{4}$ grain (0.008–0.016), preferably combined with atropin, $\frac{1}{200}$ to $\frac{1}{100}$ grain (0.00032–0.00065), which last is of value to lessen hypersecretion. Codein may be employed.

To the use of cocain for nausea and vomiting I am opposed. It gives but temporary relief, is a marked cardiac depressant, many patients have an idiosyncrasy to even a small amount, and there is danger of the cocain habit.

It may be necessary to repeat lavage several times. The belladonna or atropin should be pushed to physiologic limits, with dilatation of the pupils and dryness of the throat, if one expects to have any effect in checking the hypersecretion.

Some recommend washing the stomach with 1 : 2000 silver nitrate solution, and following it with plain warm water.

Some patients will not consent to lavage, and in these cases we can simply give internally several large doses of the alkalis with belladonna every two to three hours, whether they vomit or not.

If there is *great thirst*, a glass of hot water in which sodium bicarbonate, 1 dram (4.0), has been dissolved is of service, if taken in small amounts (teaspoonful doses). It often relieves the nausea and the alkali is of value; or a small piece of cracked ice or an occasional teaspoonful of cool water can be also employed.

The raw white of eggs beaten up, placed in a cup and packed around with ice, or the 5 to 10 per cent. gelatin, two teaspoonfuls given every half-hour or so, both relieves thirst and binds the acid; or

| | | | |
|---|---|---|---|
| Milk | ℥viij (250 c.c.); | | Pack this in ice. |
| Oxalate of cerium | gr. x (0.6); | dissolved therein. | |
| Sodium bicarbonate | gr. xv to ʒss (1.0–2.0) | | |

Give 1 teaspoonful (4.0) every half-hour, or so as to relieve thirst and bind the acid, and check nausea and vomiting.

In some cases rectal enemata of hot normal salt solution at 115° F., 1 pint (500 c.c.), relieve thirst and stimulate the pulse. It may be necessary to employ smaller quantities of saline solution or to use proctoclysis.

It is best for the patient not to drink too much the first day of the attack, except the remedies noted, as an excessive amount of fluid favors vomiting. No food should be given except as above advised. On the next day small quantities of milk diluted with lime-water or matzoon and Vichy or albumin-water can be given, $\frac{1}{2}$ to 1 ounce (16.0–32.0), every hour, and the gelatin can be kept up.

The quantity of nourishment may then gradually be increased, and, finally, soft-boiled eggs, scraped raw meat, and a subsequent diet such as is used in hyperchlorhydria.

The bowels should be moved early in the attack by a soapsuds enema, 1 quart (liter), to which 8 ounces (250 c.c.) olive oil have been added, or by enteroclysis.

To recapitulate. In the treatment of the acute attack I follow out the following method: Heat to the abdomen, the administration at once of an alkali by mouth; the bowels are moved by enema, and the stomach, after a brief period of rest, washed with an alkali, preferably with milk of magnesia; 2 drams (8.0) of milk of magnesia diluted with water, 2 ounces (125 c.c.), with tincture of belladonna, 10 drops (0.6), being poured into the stomach through the tube before removal. A hypodermic of morphin is then given, the room darkened, and the patient kept perfectly quiet. This treatment is repeated if necessary. If the patient will not consent to lavage, then alkalis, gelatin, and whites of raw eggs with belladonna should be administered.

## GASTROSUCCORRHEA (CONTINUA CHRONICA)

(*Synonyms.*—Chronic Gastrosuccorrhea; Chronic Hypersecretion; Chronic Parasecretion; Chronic Continuous Secretion of Gastric Juice; Reichmann's Disease.)

Chronic continuous secretion of the gastric juice is a perversion of function which is characterized by the secretion of an excessive quantity of gastric juice, not only after the ingestion of food, but also when *the stomach is empty.*

The chief diagnostic point is the secretion of large quantities of gastric juice in the fasting stomach. Reichmann,[1] in 1882, was the first to describe this condition.

We must make a distinction between the pure *cases of chronic* gastrosuccorrhea and those in which dilatation (atonic) of the stomach or dilatation from stenosis, especially with ulcer, exist, and with which hypersecretion is an associated symptom.

After fasting, small quantities of gastric juice, as much as 5 to 10 c.c. or 15 to 20 c.c., on one or two occasions have been aspirated. Thus the possibility of a normal small secretion may exist. If, however, we find 20 to 30 c.c. or more of gastric juice constantly in the stomach of a patient who has been fasting for some time, the finding we would consider to be pathologic and to be an evidence of continuous hypersecretion.

**Frequency.**—Cases of pure gastrosuccorrhea chronica without any associated organic lesions of the stomach I believe, with Einhorn, to be quite a rare disease, while those found in association with ectasia are not uncommon. Undoubtedly the latter class have been confused with the pure cases. Chronic hypersecretion is more rare than the periodic type.

**Etiology.**—Chronic hypersecretion occurs more frequently in men than women, and in youth and middle life.

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[1] Berlin. klin. Wochenschr., 1882, No. 40.

Severe mental strain or worry seem to be factors, also the neurotic condition.

The periodic type may develop into the chronic; persistent hyperchlorhydria or its causes, such as indigestible or irritating food or condiments, the abuse of alcohol or tobacco, or excessive hot or cold food and drinks, may be factors.

Dilatation of the stomach, ulcer, or tetany may have chronic gastrosuccorrhea associated with them.

One must also remember that chronic hypersecretion may be reflexly produced by disease of some other organ, the appendix, gall-bladder, pancreas, tuberculosis, or a new growth of the cecum, and that severe inflammation of the stomach, Fenwick[1] believes, may follow, and that even gastric ulcer or ulcer of the duodenum may result. This same hypersecretion may give rise to spasm of the pylorus, causing pain and intermittent obstruction. Fenwick even goes so far as to state that the character of the inflammation of the appendix is indicated by the gastric secretion; an active irritation being indicated by *hypersecretion*, while torsion, thickening, cystic dilatation or adhesion are followed after a time by a type of chronic gastritis characterized by flatulence, nausea, anorexia, excess of mucus, and absence of free hydrochloric acid. This last corresponds nearly to the achlorhydria gastrica hæmorrhagica described by Pilcher[2] in Mayo's clinic.

**Symptoms.**—In most of the cases the patients state that the gastric symptoms began gradually, a feeling of pressure, fulness, and sour eructation commencing a couple of hours after the ingestion of food, resembling the symptoms of hyperchlorhydria. Then pain occurs several hours after meals or shortly before the ensuing meal; it is *most frequent on the empty stomach*, just before the next meal is due. It may be spasmodic in character; nausea and then vomiting follows. The vomiting may increase in frequency, and, finally, occur several times a day after breakfast and supper. In some cases the attacks take place during the night between 12 and 2 o'clock, the patient being awakened by burning and pain in the epigastrium, acid eructations, and, finally, vomiting of very acid fluid takes place; after vomiting the pain is relieved. The night vomitus is generally a clear fluid. Albuminous food, egg or milk, will often relieve the pain. The day vomitus is very acid (quite liquid), but some food is mixed with it; is often of a grass-green color.

The appetite is generally good and often increased. Sometimes the patient develops excessive hunger; loss of appetite is rather rare.

Thirst is increased, the bowels are constipated, the urine diminished and less acid than normal.

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[1] The Clinical Significance of Gastric Hypersecretion and Its Connection with Latent Disease of the Appendix, Proc. Royal Soc. Med., April, 1910, Surg. Sect., p. 177.

[2] Jour. Amer. Med. Assoc., Nov. 19, 1910, p. 1790.

There may be some loss of weight, but no marked emaciation.

The contrast between the physical condition of the pure type of chronic hypersecretion and that in which dilatation of the stomach accompanies it will be described under Differential Diagnosis.

*Palpation.*—The stomach is more or less sensitive to pressure.

**Diagnosis.**—The diagnosis of gastrosuccorrhea continua chronica can only be made by *examination of the stomach in the fasting condition.* To exclude error, the method suggested by Reichmann is the best.

The stomach should be thoroughly washed out with warm water, preferably at night, all food and drink should be withheld for ten to twelve hours, and then aspiration of the stomach-contents should be performed. Anywhere from 50 to 125 c.c. of gastric contents are thus obtained. This secretion exhibits all the properties of the gastric juice. It contains no particles of food. The fluid is usually watery and clear in color or it may be yellowish green from admixture with bile. There is an increased degree of acidity, the values for free hydrochloric acid being, as a rule, pretty high. There are no starchy products contained therein. There are no evidences of fermentation and no organic acids present. Under the microscope no sarcinæ are found.

An hour after Ewald's test-breakfast more liquid than usual is present and the acidity is quite high (75 to 125), higher than when the fasting contents are aspirated. A thin disk of egg-albumen will be digested at blood temperature in an hour or so. Lugol's solution gives a blue or violet reaction, showing the starch is unchanged or slightly so.

Three to four hours after Riegel's test-dinner hardly any meat fibers are found, whereas considerable starchy material is present. The acid content is high and the residue considerable.

We must remember that the above description is a case of pure chronic gastrosuccorrhea in which there is no motor insufficiency. These cases I believe to be quite rare.

**Differential Diagnosis.**—Many of our writers do not properly distinguish between cases of pure chronic hypersecretion and those in which dilatation of the stomach is a complication. The examination of the stomach as regards its size, position, and motor functions should always be carried out, and this will aid us.

*Ulcer of the stomach* may be complicated by hypersecretion, and in a few rare cases gastric tetany is associated with it. Occasionally no symptoms pointing to a previous ulcer can be obtained, though there may have been a previous history of hyperchlorhydria, and at subsequent operation the stenosis will be found to be due to an ulcer not entirely healed.

In *ectasy* from pyloric benign stenosis with hypersecretion we have excessive vomiting of a large quantity of greenish-yellow fluid, pain, cramp-like attacks, peristaltic unrest, excessive thirst, skin over the abdomen dry and wrinkled, loss of weight which is often very great, and occasionally some tinge of blood in the vomitus. The patient presents almost a cachectic appearance, and there is in some patients the sense of resistance, or even a feeling of thickening at the pylorus.

The vomitus or contents aspirated after a test-meal will separate into three layers: an upper layer of foam, middle layer yellow or yellowish green, and a lower layer of sediment. Meat is digested, the sediment consists of starchy material. Acidity is markedly increased and content of hydrochloric acid is high. Pepsin digestion is rapid. Starchy materials, yeast-cells, and sarcinæ are found under the microscope.

Examination further shows marked dilatation of the stomach, with great relative motor insufficiency. *Hyperchlorhydria with atonic dilatation must also be differentiated.* The pain and vomiting present some of the symptoms of hypersecretion.

For example, in one case a test-meal given at night shows A. M. on aspiration 500 c.c. of contents; separating into three layers, fermentation being present, stomach one finger below umbilicus. Reichmann's method was then employed, *i. e.*, the stomach was thoroughly washed out and nothing given for twelve hours. Morning aspiration showed the organ to be empty, no secretion, hence the diagnosis was clear.

In many cases of marked stagnation of the stomach-contents with dilatation, continuous secretion may be simulated. The method of testing the empty stomach will determine the diagnosis.

If there have been hematemesis, melena, circumscribed tenderness, or dorsal tenderness, ulcer will be suspected. Frequent examinations for occult blood in the stomach-contents and stool are of service.

*Tetany* may complicate hypersecretion with dilatation. The symptoms are characteristic.

**Prognosis.**—In the cases of pure chronic hypersecretion the prognosis is fairly good. Most patients improve under treatment, but quite frequently there are relapses. Sometimes the condition persists for years. Hypersecretion *per se* is never fatal. When complications such as dilatation of the stomach are present, the serious features are dependent on them.

**Treatment.**—*Prophylaxis.*—The patient should not overwork, should be relieved of all mental overexertion, and lead a rational out-of-door life, with proper attention to exercise and hygiene. Nervous conditions when present should be treated. The patient should avoid bolting his food, should not eat any irritating substances, such as mustard, pepper, spices, alcohol, and very hot and very cold food and drink; in fact, he should avoid everything that will overstimulate the secretion of gastric juice. Avoid smoking.

Albuminous food is digested well, and starchy food, badly; hence the latter should be reduced in quantity.

*Diet.*—This is practically the same as in hyperchlorhydria, except that very large quantities of fluid should be avoided.

If the appetite and physical condition are good, it is just as well to give but three meals a day, so as to give the stomach a rest and not tend to keep up gastric secretion. In this event the excessive acidity can be neutralized between feedings by alkalis. Doses, $\frac{1}{2}$ to 1 dram (2.0–4.0) of magnesia usta, milk of magnesia, or soda bicarbonate should be given, as in hyperchlorhydria, in water one to two hours after meals, and the hypersecretion and the pain relieved by tincture of belladonna, 10 drops (0.6) t. i. d.

On the other hand, some patients, as in hyperchlorhydria, readily feel satiated, and yet desire food frequently. They may also be losing some weight, especially in the cases complicated with dilatation; small meals, which are readily expelled from the stomach (*i. e.*, in soluble form or mushes), must be given, and yet the nutrition must be kept up, which last necessitates frequent feeding.

The diet, for example, when given in small frequent meals would be as in hyperchlorhydria, but *less fluid:*

7.30 A. M.—Milk or cocoa, 250 c.c. (8 ounces), 2 zwieback or toast and egg (1).
10.00 A. M.—Beef sandwich or ham sandwich.
1.30 P. M.—Soup, 250 c.c. (8 ounces), with raw egg, steak (100 grams), potatoes (50 grams).
4.00 P. M.—Same as 10 A. M.
7.00 P. M.—2 eggs or meat (100 grains), 2 slices toast, butter (20 grams).

If there is dilatation, more soluble food should be given (see Dilatation of the Stomach). Starches should be given thoroughly cooked, or predigested and in small amounts, and preferably in soups and mushes; potatoes should be mashed and alcohol avoided; also avoid cabbage, turnips, spices, pickles, mustard, etc.

**Medicaments.**—*To lessen hypersecretion.*

Tincture belladonna in doses of 10 drops (0.6) or more, t. i. d. before meals, up to physiologic effects, or extract belladonna, $\frac{1}{6}$ to $\frac{1}{3}$ grain (0.01–0.02), will lessen secretion and subsequent hypersecretion. Atropin, $\frac{1}{100}$ to $\frac{1}{50}$ grain (0.0006–0.0014) t. i. d., by mouth or hypodermic, is also of value; the pain and the spasm of the pylorus and the hypersecretion are lessened by these remedies.

One can administer a large dose of belladonna after lavage before withdrawing the tube. Bismuth subnitrate, 30 grains (2.0), in 2 ounces (60.0) water, t. i. d. half an hour before meals, or olive oil, 1 ounce (30.0) t. i. d. before meals, or the latter containing 30 grains (2.0) of bismuth, lessen secretion.

Large doses of morphin, as have been recommended, I believe to be a pernicious method for obvious reasons.

*For Attacks of Pain.*—Alkalis, such as milk of magnesia (Phillips), $\frac{1}{2}$ ounce (15.0) in 2 ounces (60.0) of water, or magnesia usta, 1 dram (4.0), or soda bicarbonate alone, 1 dram (4.0), or combined with the above, are of service. Albumin-water (white of raw egg) or gelatin, 5 to 10 per cent. solution, 1 ounce (30.0), are useful. Heat should be applied externally. *Lavage, preferably* with *an alkaline solution*, with 1 ounce (30.0) milk of magnesia in 1 quart (liter) of warm water, or with soda bicarbonate, ½ ounce (15.0), or magnesia usta, ½ ounce (15.0) in the same amount of water, is the best method. A small quantity may be left in the stomach and belladonna, 10 drops (0.6), with an additional dose of the alkali poured in before removing the tube.

In some cases it may be necessary to administer a hypodermic of codein, ¼ to ½ grain (0.0016–0.032), or morphin, ⅛ to ¼ grain (0.008–0.016). They should only be given by a nurse or physician.

*Alkalis.*—If there be no ectasy, a course at Carlsbad is of service, or artificial Carlsbad salts or the imported salts, or alkaline mineral waters, such as Vichy, can be taken at meals or just before meals. This lessens hyperacidity and thus aids digestion.

To prevent subsequent attacks of pain an alkali should be given at the height of digestion, about two to two and a half hours after food; magnesia usta or milk of magnesia, ½ to 2 drams (2.0–8.0), alone or combined with soda bicarbonate or ammonia magnesia phosphate, as described under Hyperchlorhydria, are useful.

*Lavage.*—Reichmann and Riegel were the first to recommend lavage for the treatment of this condition. Reichmann and Einhorn perform lavage in the morning with the stomach fasting, while Riegel washes it out six or seven hours after the heavy meal. My method is entirely dependent on the time of appearance of the symptoms. If the attack comes on at midnight or early in the morning, it seems most logical to perform thorough lavage at bedtime with an alkaline solution, leaving some of it in the organ, and also pouring into it a large dose of belladonna, 10 to 15 drops (0.6–1.0).

If the attacks come on after breakfast or the noon meal, then the early morning lavage on the empty stomach seems best. I often recommend pouring Carlsbad salts directly through the tube before withdrawal. With *severe pain* it may be necessary to perform lavage again during the exacerbation.

If there be dilatation and marked retention of food, lavage six hours after the full noon meal is necessary, then followed by a light supper; an alkali and belladonna should be given at bedtime.

In some obstinate cases Reichmann recommends lavage with 1:1000 to 1:2000 silver nitrate solution. It is safest to wash the stomach with about 500 c.c. of this solution, 150 c.c. at a time, and then wash out the stomach with warm water. Normal salt solution may be substituted if there is much irritation. Some also recommend the internal administration of ⅙ to ¼ grain (0.011–0.016) of silver nitrate in pure form or in solution t. i. d. and at bedtime.

*Stomach Spray.*—Einhorn claims excellent results from spraying the stomach with his gastric spray with nitrate of silver 1:2000 to 1:1000 after a previous washing with warm water.

*Direct Galvanization.*—The same author reports good results from internal galvanization of the stomach, employing it and the gastric spray on alternate days. Riegel finds no benefit from intragastric galvanization unless atony is present, and in this I agree.

If the condition of chronic hypersecretion be complicated by dilatation, this condition must be treated. Rose's belt is of value for atonic ectasia. If there is pyloric stenosis, gastro-enterostomy is indicated.

Lavage with an alkali, belladonna or atropin in large doses, the alkaline treatment, diet, and in some cases spraying or lavage with nitrate of silver, are the chief requirements.

## CHAPTER XV

# DISTURBANCES OF THE MOTOR FUNCTION OF THE STOMACH—ACUTE ATONY—CHRONIC ATONY—ACUTE DILATATION OF THE STOMACH—CHRONIC DILATATION OF THE STOMACH

## ATONY OF THE STOMACH

Atony of the stomach may be defined as a relaxation and weakening of the muscular wall of the organ, so that it cannot empty itself in the normal time, and thus motor insufficiency results. It is a perversion of motor function. With simple atony, the stomach is of *normal size*, but motor insufficiency exists. When the organ is enlarged, we speak of ectasy or dilatation, which is combined with motor insufficiency.

There are two types of atony of the stomach: First, *acute atony;* second, *chronic atony*.

### Acute Atony of the Stomach

Acute atony of the stomach may occur as a preliminary to acute dilatation of the stomach, just as may chronic atony to chronic atonic dilatation.

Acute atony does not necessarily result in acute dilatation.

Many of the causes which produce acute dilatation are the factors with acute atony, but the stomach has *not become dilated*. The early recognition of the condition is, therefore, important. The motor insufficiency which occurs with it may also *lead to error in diagnosis*. The condition takes place most frequently after overloading the stomach, bolting the food, or indigestible food, or alcohol. It may complicate acute gastritis, with belching, fulness or discomfort in the stomach, some distention, constipation, and *delayed vomiting;* in fact, merely discomfort for a considerable period of time and then the *vomiting of food taken some hours before*. The splashing sound is present. The stomach becomes distended, tympanitic, and often sensitive to pressure. A sudden attack of retention of chyme for an abnormal length of time is the salient symptom.

In one case, because of motor insufficiency following a single dietary indiscretion, a diagnosis of ectasy with motor insufficiency was made. Examination demonstrated normal functions, the attack evidently being an acute atony (acute motor insufficiency) of temporary duration.

During typhoid or the infectious diseases, gastric disturbances with belching, discomfort, sudden *distention of the stomach*, constipation, or diarrhea, with *delayed vomiting*, the vomitus consisting of milk (curdled) or *other food taken some hours previously*, are significant of this condition. There may be evidences in *the stool* of nourishment *taken forty-eight hours before.*

**Treatment.**—The immediate emptying of the gastro-intestinal tract is the indication. *Lavage should be performed at once.*

A good cathartic, calomel, 3 to 5 grains (0.2–0.3), or blue mass, 5 grains (0.3), followed by a saline cathartic, should be given.

Castor oil, 1 to 2 ounces (30.0–60.0), is also excellent. Enemata, and especially rectal irrigation, are of value to produce intestinal peristalsis.

Acute atony may progress rapidly to acute dilatation or develop more slowly for ten to twelve hours, and then, if untreated, result in acute ectasy. Its early recognition is, therefore, important.

## CHRONIC ATONY OF THE STOMACH

(*Synonyms.*—Motor Insufficiency; Gastric Insufficiency; Motor Insufficiency of the First Degree; Myasthenia Ventriculi; Atonia Gastrica.[1])

Chronic atony of the stomach, if untreated, may result in the atonic type of dilatation of the stomach.

Its correction is, therefore, of great importance. We use the term " chronic " in distinction from the acute, evanescent form. In the *pure cases no dilatation is present.*

**Etiology.**—Atony of the stomach may complicate many digestive disorders, such as chronic gastritis, hyperchlorhydria, neurasthenia gastrica, and diseases of the heart and lungs, as tuberculosis. It occurs in nervous and hysteric subjects and may exist as a *primary neurosis.*

It may occur as a result of biliary colic or the crisis of tabes.

**Symptoms.**—If atony occurs as a complication of some other affection of the stomach, its symptoms will be overshadowed by the primary disease. The characteristic symptoms of atony are: a feeling of fulness after meals, slight distention, belching of gas, diminution of appetite, headache, and constipation. The resulting motor insufficiency is productive of fermentation and gas production.

**Physical Examination.**—There is generally some distention of the stomach with gas. The splashing sound is easily produced over the greater part of the stomach an hour or two after the test-breakfast, or four or five hours after a full meal. If the splash extend to the *umbilicus* or *below it*, this is evidence that *dilatation* is associated.

If movable kidney is associated, gastroptosis is present. These are not pure cases.

An hour after Ewald's test-breakfast aspiration of the stomach-contents will remove 100 c.c. or more of gastric contents; an excellent test taken in connection with the symptoms.

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[1] Rose has called to our attention that atonia gastrica is an improper term, really meaning abdominal relaxation. It is so applied in our work to define splanchnoptosis.

Six hours after Leube's test-dinner, aspiration and lavage show considerable chyme, 150 to 200 c.c. The fasting stomach in the morning is found empty.

Boas states that on filling the stomach with water, the greater curvature will descend as water is added. This is not reliable. The lower border will descend after a moderate amount of water, if the stomach is dilated.

**Prognosis.**—This is good if proper treatment is instituted.

**Treatment.**—If hyperchlorhydria or chronic gastritis is present, each should receive appropriate treatment; as should tuberculosis, endocarditis, nervous conditions, etc.

In all cases of chronic atony of the stomach, Rose's plaster belt is indicated. Atony of the intestines is frequently associated, and the use of mechanic support lends tone to the general musculature of the abdomen, keeps the stomach well supported, and prevents *atonic dilatation of the organ.*

*Vibratory massage* or massage over the entire gastro-intestinal tract is of value. It stimulates the muscles and lessens the tendency to constipation. Outdoor exercise, walking, golf, and horseback riding are indicated. In some cases douching the abdomen is of service; in this event one must employ a silk elastic abdominal support, which can be readily removed. I have secured the best results with Rose's belt. The patient should not overwork, either mentally or physically, and should eat slowly and masticate the food thoroughly; the teeth should be kept in good condition. An excessive quantity of fluid should not be taken—in all, including water, soups, tea, etc., not over $1\frac{1}{2}$ liters a day.

It is best to give numerous divided meals of rather moderate size, four or even five daily, so as not to overburden the stomach with three large meals, and allow it to thoroughly empty itself.

The diet may include bread (fresh and hot breads are interdicted) and butter, eggs in various forms, cereals, milk, soup, chicken, steak, chops, game, squab, fish, oysters, and green vegetables, which are specially valuable for the constipation; cocoa, weak tea, occasionally weak coffee, with milk and sugar. The diet must be modified if hyperchlorhydria or chronic gastritis is present, or to suit the special idiosyncrasies of the patient. Alcohol should be interdicted. An occasional cigar smoked with a holder is allowable unless there is catarrh.

*Medicaments.*—Strychnin or nux vomica is of great service to tone up the muscular system. They may be given alone, or

℞. Tr. nucis vomicæ........................ ♏x (0.59 c.c.);
Comp. tinct. cinchona.................... ♏xv (0.888 c.c.).—M.

Sig.—Give in a wineglassful of water t. i. d. half an hour before meals. Some prefer it at the same time after meals.

or, if the patient is anemic, it may be combined with iron and arsenic, thus:

℞. Strych. sulph. . . . . . . . . . . . . . . . . . . . . . . . . . . . . . gr. $\frac{1}{30}$ (0.0021);
Sod. arsen. . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . gr. $\frac{1}{50}$ (0.0013);
Blaud's iron pill . . . . . . . . . . . . . . . . . . . . . . . . . . gr. v (0.3).—M.
One pill; administer t. i. d. after meals.

Strychnin sulphate, $\frac{1}{60}$ to $\frac{1}{30}$ grain (0.00108–0.021), is excellent, or tincture nux vomicæ in combination with fluidextract of condurango, or compound tincture of cinchona, thus:

℞. Tinct. nucis vomicæ,
Fluidext. condurango } . . . . . . . . . . . āā 125 c.c. (about ʒij).—M.
Dose, 20 drops in water, t. i. d. before meals.

If hyperchlorhydria is present, I omit strychnin; though Musser advocates nux in the neurotic type.

Other useful iron preparations are:

Iron tropon . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . ʒj to ij (4.0–8.0), t. i. d.
Peptomangan (Gude) . . . . . . . . . . . . . . . . . . . . . . . ʒj to iij (4.0–12.0), t. i. d.
To be given in water or milk after meals.

*Electricity.*—This is of service applied by the external method or, in some cases, by intragastric faradization.

Static electricity or the high-frequency current may be useful in nervous cases.

*Lavage* is not indicated.

*Massage* or *vibratory massage* is especially useful.

*For Constipation.*—The green vegetables, brown, rye, and Graham bread, and raw or stewed fruits are serviceable. A glass of water should be taken on rising. The patient should accustom himself to go to stool at a definite hour, and on the closet may gently massage the stomach and bowels to aid action.

A small gluten or glycerin suppository, or the injection of 1 ounce (30.0) of olive oil, or 4 to 6 ounces (125–185 c.c.) of warm water have a good effect in exciting peristalsis; or a soapsuds enema, but never of larger size than 1 quart (liter). Olive oil injection, 1 pint to 1 quart (500 c.c. to 1 liter), at bedtime, to be retained, is useful.

It may be necessary to employ medication, such as extract of cascara, 1 to 4 grains (0.065–0.26), at bedtime, or fluidextract of cascara, 1 to 2 drams (4.0–8.0), or pills, such as the lapactic, aloin, belladonna, podophyllin, phenolax, purgen tablets, regulin, etc.

## ACUTE DILATATION OF THE STOMACH

Acute dilatation of the stomach may be defined as acute atony of the stomach, with resulting acute motor insufficiency, gradually merging into a paralytic condition, and accompanied by a distention of the organ to beyond its normal physiologic limits. Its lower border extends to the umbilicus or usually to below this point, and the stomach may even occupy the entire abdominal cavity.

Brunton, Fagge, Boas, Hemmeter, notably Campbell Thomson,[1] and Lewis A. Conner,[2] have written on this subject. The fatal or most severe cases have been reported, but the condition occurs quite frequently. I have already referred to numerous types.[3] Personal investigation demonstrates that five anatomic types of acute ectasy exist:

1. Acute dilatation of the stomach alone.
2. Acute ectasy, which supervenes on an existing chronic dilatation (due to stenosis of the pylorus). Thomson also reports one case.
3. Acute dilatation of the stomach and duodenum, the most fatal type.
4. Acute dilatation of the stomach engrafted on chronic atonic dilatation.
5. Acute dilatation of the stomach and intestines, a mixed type, so-called acute tympanites.

This last is quite common, especially in the acute infectious diseases, such as typhoid fever and pneumonia.

For a complete description of the theories of its mechanism and of the subject, I refer my readers to the "American Journal of Surgery," November–December, 1908. Weston and the author found that the fibrous attachment of the transverse duodenum to the diaphragm (muscle of Treitz) and the pressure of the dilated stomach on the transverse duodenum were chief factors in the production of the gastroduodenal type of dilatation. Mesenteric traction was chiefly produced by the downward pressure of the stomach on the intestines, which last exercised a countertraction against the muscle of Treitz. The collapsed intestines were the result of pressure.

**Mechanism of the Production of Acute Dilatation of the Stomach.**—The nature of the condition as stated is undoubtedly an *acute atony*, with acute *motor insufficiency*, finally merging *into a paralytic condition*. Many factors in the production of acute dilatation have been described, namely:

1. Section of the vagi—by Carion and Hallion—producing acute dilatation of the stomach, thus demonstrating that an injury or inflammation of these nerves may be a cause, as in cerebral injury, or pneumonia at the base.
2. Injury to the dorsal spine by stimulation of the inhibitory nerves.
3. Direct action of the agent on the musculature or its terminal nerve-filaments; among such may be chloroform, anesthesia, toxemia from fermentation, etc.
4. Traumatism.

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[1] Brochure.

[2] Amer. Jour. Med. Sci., March, 1907.

[3] Med. News, Aug. 6, 1904.

5. Spasmodic stenosis of the pylorus, due to fermentation or hyperacidity.

6. Acute gastrorrhea (Morris); or possibly acute gastrosuccorrhea (Kemp).

7. Kelling's and Conner's experiments on spasmodic closure of the cardia, and the demonstration of kinks in various parts of the duodenum, or of spasm of the pylorus.

8. Rotation of the pylorus.

9. Kelling's and Braun's experiments, demonstrating that acute dilatation of the stomach is a paralytic condition by producing it with animals under deep narcosis.

10. Rotation at junction of cardia and esophagus.

11. Toxemia from infection, as from the toxins of typhoid, pneumonia, etc.; or auto-intoxication from improper diet, causing gastro-intestinal dilatation during the course of these diseases. Dietary indiscretions are the causes of acute ectasy of the milder types which I shall shortly describe.

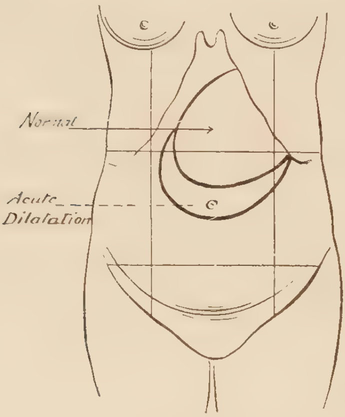

Fig. 172.—Experiment: Acute dilatation by $CO_2$ distention.

12. *Obstruction of the transverse duodenum* is one of the frequent causes so far found in the fatal cases, producing acute gastroduodenal dilatation, the most dangerous type. There are a number of causes given for this condition, notably:

(*a*) Mesenteric obstruction of the duodenum from mesenteric traction. Out of 69 fatal cases, 19, or 27.5 per cent., Conner states were caused by this, and probably 33 to 50 per cent. is nearer the figure, according to his view. Albrecht first called attention to its condition, performing numerous experiments.

(*b*) Pressure from the distended stomach on the transverse duodenum, producing complete obstruction, is most frequently the cause in my belief.

(*c*) The firm fibrous band (muscle of Treitz) attaching the transverse duodenum to the crus of the diaphragm, a factor hereafter noted and demonstrated by Dr. Weston and myself, is also an important factor.

Regarding mesenteric traction, Conner further believed that the conditions essential for this were the dorsal position, an empty intestine, and a mesentery of such length that the intestine can slip into the pelvis and yet hang free. He holds that fasting, purges, and enemas after operation have a possible bearing.

Accessory factors are suggested, such as a lax abdominal wall, pressure from weight of hepatized lungs (in pneumonia), coughing and laughing paroxysms, lordosis, or an abnormal position of the duodenum. It generally crosses the third lumbar vertebra; the fourth is the most prominent, and more pressure would be exercised at this point.

Clotted blood behind the transverse duodenum was a cause in one case.

On the other hand, some believe the stomach dilatation is the primary factor, and the mesenteric constriction is produced secondarily by the stomach forcing down the intestines.

T. Satterthwaite and the author studied the effects of acute gastric distention on the pulse and respiration by artificially distending the stomachs of patients with carbonic acid gas and taking the blood-pressure before, during, and after the experiment. In Fig. 172 is illustrated the result in one of our cases.

Physician.....................Position dorsal.

| | Pulse. | Pressure. | Respiration. | Pulse. |
|---|---|---|---|---|
| Before distention........... | 72 | 135 | 18 | Slightly irritable (tobacco). |
| After distention............ | 86 | 120 | 24 | Irregular in force; some intermission in the beats. |

Pain under the sternum and over the abdomen, sensation of suffocation and of flushing of the face, nausea, and discomfort accompany the acute distention. The stomach extends to one fingerbreadth below the umbilicus; in all a distention of $3\frac{1}{2}$ fingers-breadth.

Aspiration of the contents relieved the symptoms. These facts emphasize the danger of overdistention of the stomach with carbonic acid gas for testing the position of the organ in patients with cardiac or pulmonary disease, or in old age, also the danger of acute ectasy as a complication. Moreover, the true dimensions of the stomach may not be obtained by the method of carbonic acid gas distention.

In this connection, the author feels that he must briefly refer to an article[1] entitled "The Non-entity of Acute Dilatation of the Stomach," to which some physicians have referred. As a sample the writer states, "But as has been already intimated, nothing short of crushing and stretching of the musculature should cause acute dilatation, and such causes do not exist." Such productions, when taken seriously by the profession, do positive harm. The author invites the writer of this article or any skeptic to test on himself the experiment with tartaric acid and bicarbonate of soda. Ingest separately about 3 drams (12.0) each of soda bicarbonate and tartaric acid in 8 ounces (250 c.c.) of water and observe the results. A physician or nurse should be at hand with a stomach-tube.

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[1] Med. Rec., Nov. 5, 1910.

**Etiology of Acute Ectasy.**—It may be primary or secondary. The causes are as follows: Indigestible food; infectious diseases, such as typhoid, pneumonia, acute tuberculosis, and scarlatina; during convalescence from long-continued disease, as chronic tuberculosis, hip-disease, pneumonia, typhoid, sarcoma, and anemia; injury to the head or spine; traumatism to the abdomen; postoperative, in which manipulation of the viscera, shock, uremia, sepsis, and the anesthetic are factors; one case after gastro-enterostomy reported by the author; retroperitoneal abscess; disease and deformity of the spine, lordosis,

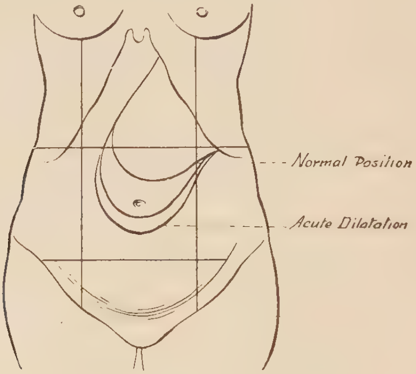

Fig. 173.—Acute dilatation of the stomach during epileptic attack.

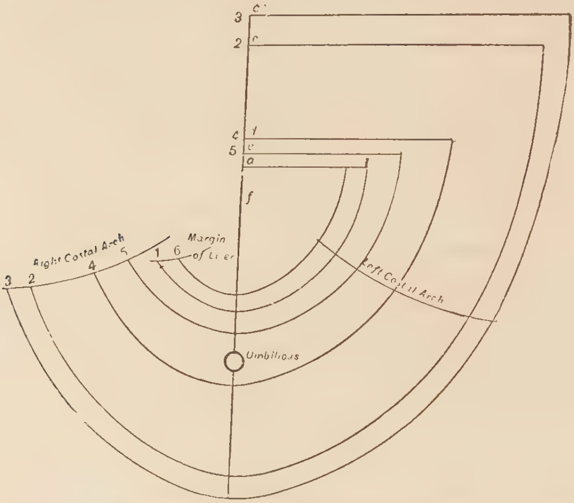

Fig. 174.—Acute dilatation of the stomach in migraine, August 29, 1902. Borders of stomach: 1, August 16; 2, August 29, in the morning; 3, August 29, in the evening; 4, August 30, in the morning; 5, August 30, in the evening; 6, August 31, in the morning.

etc.; application of plaster jacker in spinal deformity; paroxysm of laughing supposedly (true cause undiscovered). Toxemia or auto-intoxication are, therefore, factors in many cases.

*Age.*—It may occur from infancy to old age. Three-fourths of the cases are developed during adolescence or early adult life (ten to forty years).

*Sex.*—Is about equally divided.

Clinically, we may classify acute dilatation of the stomach into cases presenting various clinical types, in this sense atypic, and into the typic cases, which are usually described.

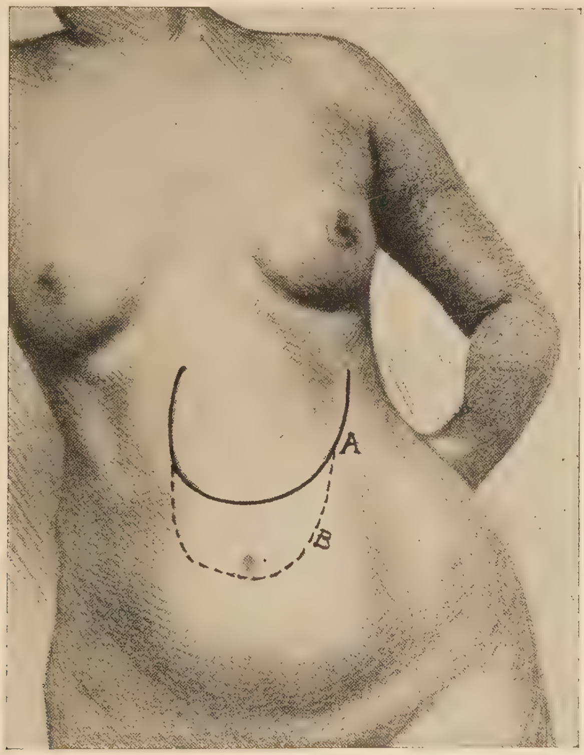

Fig. 175.—Acute dilatation of stomach with tachycardia.

**Clinical Types of Acute Ectasy.**—I shall briefly refer to these cases, which have already been fully reported by me.[1]

1. *Cases of Acute Dilatation of the Stomach with Symptoms Pointing to the Nervous System.—Convulsions in Infants and Young Children.*—Auto-intoxication is the cause. I have seen a case in an infant two years of age in which the stomach extended 2 inches below the umbilicus. Vomiting of bread and curds occurred, with immediate

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[1] Amer. Jour. of Surg., Nov.–Dec., 1908.

cessation of convulsions and return of the stomach to normal position. Repeated attacks may lead to chronic ectasy or epilepsy.

*Epilepsy.*—Mangelsdorf[1] has demonstrated acute ectasy during the convulsive seizure and the gradual return to normal position (Fig. 173).

*Migraine.*—The same author noted similar conditions during attacks of migraine (Fig. 174), and reports 500 *cases in epilepsy and migraine.*

Lauder Brunton[2] noted transitory dilatation in sick headache.

*Tetany.*—Broadbent[3] describes a case of *acute ectasy with tetany*, which ended in recovery.

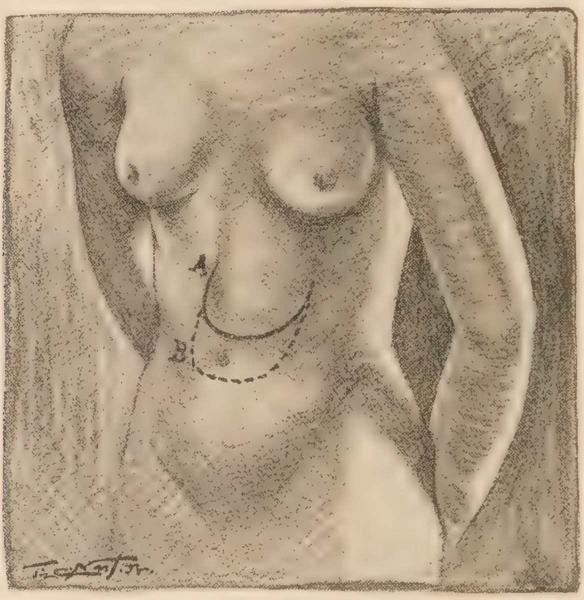

Fig. 176.—Acute dilatation of the stomach with symptoms of pseudo-angina pectoris.

*Chorea.*—Acute ectasy has been reported as a terminal event in chorea.[4]

3. *Acute Ectasy Producing Acute Cardiac Symptoms.—Tachycardia.*—Girl, age twenty-one, with chronic endocarditis, excellent compensation, no gastric disturbances. Tachycardia, 210 beats per minute, followed dietary indiscretion. Acute ectasy was found as in Fig. 175. Emesis occurred, the stomach contracted to normal size, and the tachycardia ceased. The patient suffered from several attacks, but has had no further trouble since she has exercised care in diet.

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[1] Rose and Kemp, Atonia Gastrica.

[2] Allbutt's System of Medicine, vol. iii, p. 392.

[3] Practitioner, 1908.

[4] Lancet, April 19, 1890.

*Pseudo-angina Pectoris.*—Female, age sixty-five, suffered from attacks of pseudo-angina, following dietary indiscretions, at times consciousness was lost. A number of attacks occurred and in every instance acute ectasy was present, as in Fig. 176. Emesis relieved both the dilatation and the attack. Ultimate cure resulted from proper diet.

4. *Acute Ectasy Complicating Infectious Diseases, Notably Typhoid and Pneumonia.*—The tympanites of typhoid is frequently not purely intestinal. Acute gastro-intestinal dilatation is by no means rare; in fact, I have found it quite frequent. Systemic infection or improper diet cause this condition. Acute gastroduodenal dilatation has been reported.

In the milder types of acute gastro-intestinal dilatation there is often *no vomiting.* There are cardiorespiratory symptoms which might suggest pulmonary involvement. Examination shows acute distention. Postural treatment,[1] by elevation of the head of the bed, enteroclysis, and lavage, will relieve the symptoms. The same mixed type of distention may occur with pain and shock and simulate perforation. After relief of the distention, examination shows *absence of muscular rigidity* (no peritonitis).

In the acute distention of typhoid, with intestinal hemorrhage, *lavage will relieve gastric distention* and *diminish intra-abdominal tension.* I have never seen it recommended.

*Pneumonia.*—We may have the gastroduodenal type of acute ectasy, of which several cases are reported. The mixed type is quite common and constitutes a serious danger. Undoubtedly, sudden heart-failure has been precipitated by this condition. The etiology and treatment are the same as in typhoid fever. There is greater danger to the heart from the pressure than from the passage of the stomach-tube.

5. *Acute Ectasy with Coprostasis the Prominent Symptom.*—Male, age fifty-five, following indiscretions in diet, complained chiefly of coprostasis, with occasional vomiting. Constipation had been present eight days when I saw the case. Fecal impaction was present in the sigmoid and caput coli.

Frequent lavage, enteroclysis, and cathartics relieved the condition. In Fig. 177 is depicted the stomach before and after relief.

6. *Acute Dilatation Supervening on Chronic Ectasy.*—Female, age forty-five, suffered from chronic ectasy due to pyloric stenosis from ulcer. Following a dietary indiscretion, acute ectasy resulted with gastrosuccorrhea, pain, vomiting, peristaltic waves, constipation, and collapse.

In Fig. 178 is shown the acute ectasy, with return to the position of chronic ectasy after lavage. Subsequently gastro-enterostomy was performed with a gain of 100 pounds in the patient's weight.

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[1] See Treatment of Typhoid Fever.

**Symptoms of the Severe Cases.**—The symptoms are characteristic, and are as follows:

Sudden abdominal distention, pain, tenderness, excessive vomiting, constipation, thirst, scanty urine, and collapse. It has been mistaken for intestinal obstruction or for peritonitis.

*Onset.*—This is nearly always sudden. The patient may be well or suffer from some illness taking its usual course, when he suddenly complains of great distention, discomfort, or severe pain in the abdomen. This is rapidly followed by vomiting, which is the most constant symptom, begins early, and generally persists throughout the attack. Rarely there may be an intermission due to temporary cessation of secretion, or cessation of vomiting may be a terminal event, the abdominal muscles and diaphragm being no longer able to expel the contents. Cessation of vomiting is, therefore, not always a favorable symptom.

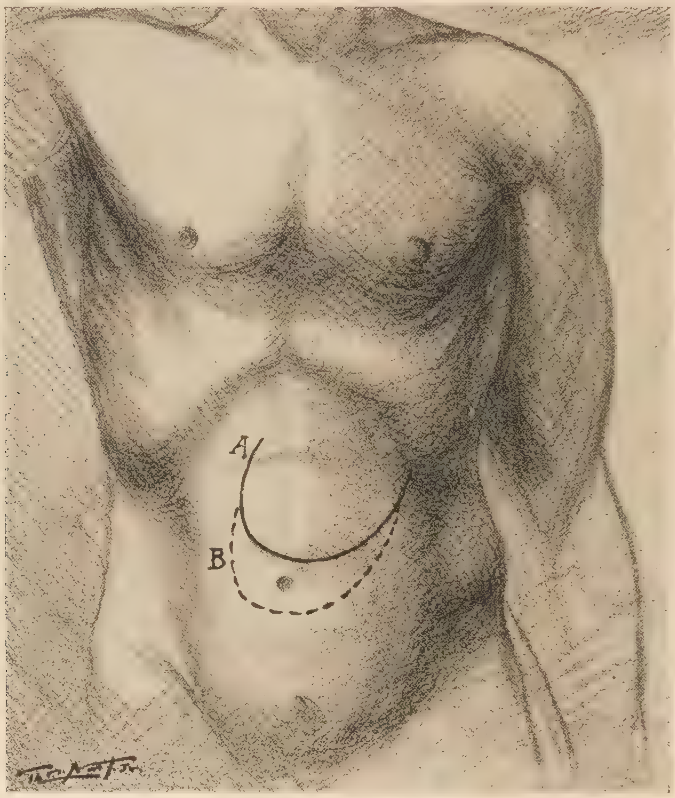

Fig. 177.—Acute dilatation of the stomach, with constipation (ten days' duration) a prominent symptom.

The vomiting is profuse, in large amounts, and comes up in gulps without straining. In the early stage it may consist of the gastric contents, of food in various degrees of fermentation; later, it becomes thinner and watery, and generally of a greenish hue. It is often described as bilious. It may be brownish, grayish, or even inky black; occasionally there may be a trace of blood. Often in the postoperative cases acute distention, pain, and greenish vomiting are the first symptoms. The vomitus may be sour or foul or even (rarely) feculent in odor.

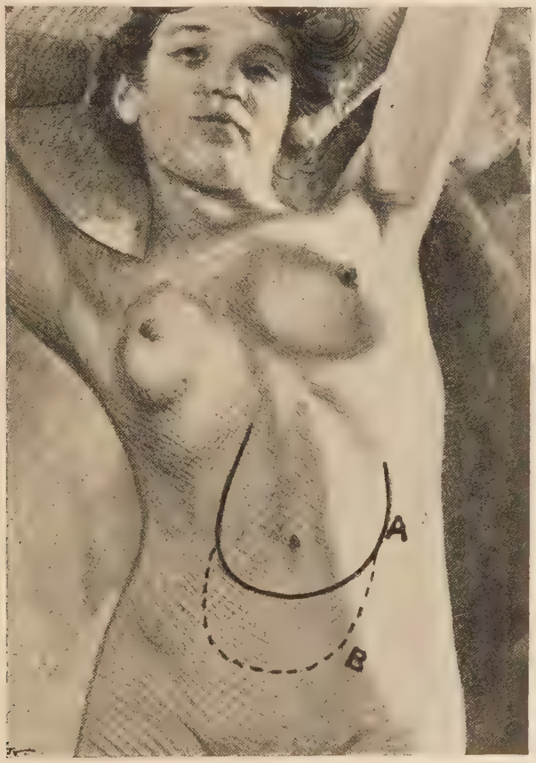

Fig. 178.—Acute dilatation of the stomach engrafted on chronic dilatation.

*Character of the Vomitus.*—Various constituents, such as bile, diastase ferment, hydrochloric acid, lactic acid, and traces of blood, visible or occult, have been found.

*Pain* is present in the majority of cases, usually in the epigastric and umbilical regions. In the gastro-intestinal mixed cases at the commencement it is more general and acute from sudden distention, being suggestive of peritonitis. Sudden perforation may even be suspected. Later there is a feeling of distention, not so acute, with accompanying cardiorespiratory symptoms. It differs from the continuous pain of acute obstruction.

*Tenderness* occurs in some cases. *Muscular rigidity is absent.*

The urine becomes scanty and nearly suppressed during the last twenty-four hours. Anuria is diagnostic of obstruction high up in the intestinal tract, and does not occur with obstruction of the large intestine. It has been mistaken for uremia. The temperature is usually normal or subnormal, unless the patient has preceding fever. Thirst is marked. Hiccough may occur as a terminal symptom, as may also delirium. General muscular cramps occurred in one case; and Broadbent reports a case of tetany.

*Physical Signs.*—In the gastric or the gastroduodenal type there is distention of the abdomen, but the swelling is not uniform; it chiefly fills the left half and lower part of the abdomen, and the right hypochondrium appears to be flattened. There is often swelling in the epigastrium.

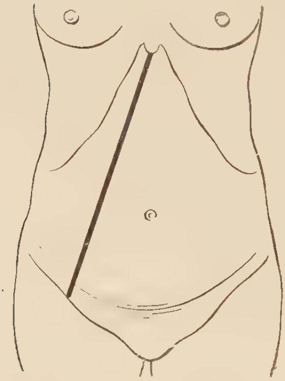

Fig. 179.—Line drawn from ensiform to Poupart's ligament.

The following is of service: Draw a line from the tip of the ensiform to the junction of the middle and outer third of Poupart's ligament (Fig. 179). The distention usually lies to the left and below this oblique line. Occasionally it appears more below the navel and sometimes there is general distention.

*Splashing sounds* (*succussion*) and the sense of fluctuation are an aid in some cases. They are not always present in the early period, when there is chiefly gas in the organ. They occur below the level of the umbilicus.

*Percussion* will show the resonance increased, but will be interfered with when there is much fluid. It is important when the splash is absent.

*Peristaltic waves of contraction occur very seldom.* They are found only before complete paresis takes place, or in the acute cases engrafted on the stenotic type of chronic dilatation.

The *general symptoms* are those of collapse, a rapid and small pulse, frequent respiration, a clammy skin, and subnormal temperature.

*Duration of the Attacks.*—The duration of the attack depends on its severity and type of case. In my case of tachycardia it lasted less than an hour. In the mixed cases it depends on the treatment accorded by the physician.

Among the *severe cases*, 1 case of Conner's died within three hours after the onset of the pain, which, with distention, was the first symptom. There was no vomiting. Several cases died within twenty-four hours, and sixteen days was the longest duration. The average was about five days. Some cases recur or several weeks may elapse, before the dilatation entirely disappears.

**Prognosis.**—In the severe cases it has been extremely bad, being a most formidable condition. There is a 72 per cent. death-rate recorded. In reality many cases occur, and with our present knowledge the mortality should be comparatively small.

**Morbid Anatomy.**—The postmortem appearance of the stomach is quite characteristic, being cylindric and bent into a horseshoe or V shape. The cardiac portion is the longer (Fig. 180).

The walls of the stomach are distended and thinned. A large amount of elasticity is retained, as shown by the shrinking that takes place after the distending force is removed.[1] The stomach may occupy the entire abdomen or even reach into the pelvis. It has been mistaken on operation for cyst of the pancreas. Its color may be purplish red, gray, or bluish white.

*Microscopic Examination.*—This has been made in a few cases. There was thinning of the musculature and no definite microscopic changes were noted, though in some there were small hemorrhages.

*Duodenum.*—In a large number of cases—over 50 per cent. (Conner)—a part or the whole of the duodenum shared in the dilatation; in many the distention stopping where the mesentery crossed. Kinks have also been found in the duodenum. The coils of the intestines are flattened and collapsed in the pelvis.

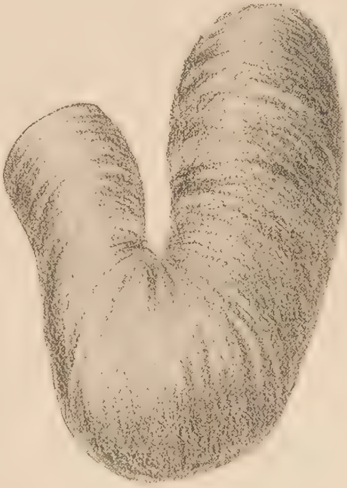

Fig. 180.—Postmortem appearance of acute dilatation of the stomach.

**Diagnosis.**—One should always think of the possibility that tachycardia, convulsions, epilepsy, migraine, anginoid symptoms, increased cardiac and respiratory rapidity, and even tetany may occur with acute ectasy. Sudden gastro-intestinal dilatation is quite frequent in typhoid and pneumonia. Often vomiting is absent in the milder cases, and the chief symptom pointing to the abdomen is distention. At times there may be acute pain in typhoid, simulating perforation. Examination in every case should be made *by percussion, the splashing sound, and especially by lavage, if there is any doubt.*

Intestinal irrigation (recurrent) will often relieve distention when present, and aid in diagnosis.

*Postnarcotic vomiting* may become gradually persistent, and this should be looked on with suspicion.

Pain, tenderness, distention, continuous vomiting of bilious appearance are suggestive, especially with collapse, rapid and feeble pulse, *urinary suppression,* and obstinate constipation.

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[1] McEvitt, N. Y. State Jour. of Med., July, 1906.

In the severe cases there may be occasionally no vomiting and no pain, but only acute distention and some diarrhea. The use of the stomach-tube and determining the position of the *stomach before and after its employment* are of value in all cases.

*If, after lavage, the distention disappears and there is no distention of the intestines*, or only slight tympanites in the colon, the acute dilatation is of the stomach or of the gastroduodenal type.

If intestinal distention persists, the dilatation is of the mixed type.

If all the tympanites is relieved by enteroclysis, subsequent examination will determine the stomach not to be dilated.

The presence of pancreatic juice, absence of fecal vomiting, and presence of bile show the gastroduodenal type. This is especially true if the abdominal posture affords relief. Bile, however, may be present, with acute gastric distention alone.

True stercoral vomiting shows obstruction lower down, and *muscular rigidity* is a symptom of peritonitis.

**Treatment.**—*Prophylaxis.*—Rapidity in operating, the minimum amount of manipulation of the viscera, a minimum quantity of anesthesia, and care in feeding after operation are important. Distention following operation should receive *immediate treatment by lavage* and *enteroclysis*. These methods will *prevent subsequent* acute *dilatation of the stomach* and should be *advocated in all cases.*

The abolition of milk-diet in typhoid fever, pneumonia, and acute infectious diseases is advisable, with the substitution of broths, strained soups of various kinds, as barley, rice, and gruels. If the patient has had an attack, then the conditions of the gastric secretion should be investigated, irregularities corrected, and proper diet instituted.

In *every case* of acute ectasy the stomach should be immediately evacuated by lavage. It is an error to wait until the symptoms appear marked, or until the patient vomits, before lavage is instituted. The stomach may redistend in the severe cases.

It is advisable to repeat lavage within two hours or at times in three hours, and thereafter every four to six hours, during the first twenty-four hours, depending on the physical signs and symptoms. It may be necessary to perform it more frequently. At times it must be carried out for some days. It is safer *to err on the side of frequency.*

*No food or drink should be given by mouth.* For severe thirst, saline enemata, proctoclysis, or even hypodermoclysis may be administered. They are also efficient in the collapse, and infusion may be required. Rectal feeding must be kept up for several days until symptoms disappear.

If there is intestinal distention, continuous rectal irrigation with normal saline solution at 120° F. is of value. It is advisable to promote peristalsis as soon as possible. Unless *hemorrhage*, peritonitis, or appendicitis complicate (as might occur in typhoid), *or there be a sus-*

*picion of a true intestinal obstruction*, after washing the stomach with plain water, in which milk of magnesia, 2 ounces (60.0), has been dissolved, I give calomel, 3 to 5 grains (0.2–0.3), in water, $\frac{1}{2}$ ounce (15.0), directly through the stomach-tube before removal; and a saline cathartic by the same method four to six hours later.

In some cases I have given by preference a high enema of 4 ounces (125 c.c.) of a saturated solution of magnesium sulphate two hours after lavage. This may be preferable, lest the patient vomit the saline cathartic.

Tincture of belladonna is useful. It lessens the secretion, relaxes pyloric spasm, and has an excellent effect on the atony. It should be given in 5- to 10-minim (0.296–0.592) doses on the tongue, with strychnin, $\frac{1}{60}$ to $\frac{1}{30}$ grain (0.00108–0.00212), every four to six hours by hypodermic injection. The latter stimulates the musculature, the heart, and respiration. Atropin, $\frac{1}{100}$ grain (0.00065), may be substituted hypodermically for belladonna.

Physostigmin sulphate (eserin), $\frac{1}{100}$ grain (0.00065), has been recommended to promote evacuation of the bowel. I have recently employed $\frac{1}{50}$ grain (0.0013) every two hours for several doses with success. It is well to arrange to give strychnin, $\frac{1}{100}$ to $\frac{1}{60}$ grain (0.00065–0.00108), to guard the eserin. Hormonal[1] (peristaltic hormone) has been suggested, by intramuscular or, preferably, by intravenous injection, for the paretic condition of the intestines. Average dose, 20 c.c.

Rectal electric irrigations of the bowels are efficacious for obstinate constipation.

The second most important therapeutic measure is *postural treatment*. The position depends on the anatomic type of the dilatation.

(1) The semi-oblique or nearly sitting position, *the head of the bed is blocked up* as in the illustration under Typhoid Fever. The patient lies on an inclined plane. This is of value in the *acute gastro-intestinal* (or mixed) *type*, with general abdominal distention, so frequent in typhoid or pneumonia, where the cardiac and respiratory symptoms are pronounced *from abdominal pressure*. By elevation of the head of the bed in one severe case of typhoid the tympanitic area in the thorax lowered 4 inches, and the pulse and respiration dropped 20 points each.

A fatal issue may result from pressure-effects on the heart and lungs with this type.

Frequent lavage and enteroclysis should be instituted in these cases, and later milk-free diet, substituting soups and broths.

This position would be *incorrect* in the gastroduodenal type of dilatation.

(2) Elevation of the foot of the bed to relieve pressure on the duodenum. The objectionable feature is *the danger from pressure on heart and lungs* if the stomach should begin to redilate.

[1] Medizin Klinik, 1910, No. 11.

(3) In the acute gastroduodenal type the *lateral position*, on the right or left side, has relieved the symptoms. The patient recovered.

(4) The abdominal position (*patient lying on the belly*) is *the best method* to treat the acute gastroduodenal type.

That this position affords relief seems to me to show quite conclusively that this type of obstruction is caused chiefly by the stomach pressure on the transverse duodenum.

Baumler kept the patient fifteeen minutes in the knee-chest position in each two hours; the balance of the time on the belly.

*Operations.*—These have not generally proved successful. Among those performed or suggested were:

The stomach opened and evacuated, and gastro-enterostomy. Gastric fistula might be tried. A kink at the duodenojejunal junction was relieved in one case, and the patient recovered.

Recovery has also been reported after one case of gastro-enterostomy.

Frequent lavage, combined with postural treatment, enteroclysis, and securing bowel action as soon as possible are indicated. Food and drink should be interdicted. Relieve thirst by enemata of saline solution and by proctoclysis.

## CHRONIC DILATATION OF THE STOMACH

(*Synonyms.*—Ectasy; Ectasia Ventriculi; Gastrectasy; Ischochymia—Einhorn; Motor Insufficiency of the Second Degree—Boas.)

The term " dilatation of the stomach " is employed for descriptive purposes, but in view of the existence of an acute type of dilatation of the organ, chronic dilatation of the stomach would seem a preferable nomenclature.

**Definition.**—How may dilatation of the stomach be defined? Is it to be measured by the capacity of the stomach alone or by the increased capacity plus the alteration of its functions? The latter is correct. The capacity of the normal stomach is extremely variable. Ziemssen has shown that a stomach may be normal and only contain 8 ounces (250 c.c.), whereas another stomach, also normal, may possess a capacity of 2 quarts (liters). The large stomach (" megalogastria "), at times found during a physical examination, produces no symptoms. This may be congenital or acquired by large eaters or by those who live on vegetable diet. Such cases, however, can readily develop atony. As long, however, as the functions of the stomach are normal, we cannot regard the conditions met with as pathologic, and hence cannot consider that dilatation exists.

As already described, the lower border of the normal stomach when distended with food or liquid lies from 1½ to 2 fingers-breadth above the level of the umbilicus. Examination of normal subjects, complaining of *no symptoms*, will frequently show that the stomachs are abnormally large, or in an abnormal position. Such cannot be considered pathologic. If it descends to nearly the level of the umbilicus, to its level or below it, and symptoms accompany it, we must consider the organ dilated.

One must not commit the error of mistaking gastroptosis for dilatation. With gastroptosis the upper border of the stomach descends as well as the lower border, and there are *movable kidney* and enteroptosis. There are varying degrees of gastroptosis.

The prolapsed stomach may, in addition, be dilated. With dilatation the upper border of the stomach does not descend, but maintains its relation to the diaphragm, and the stomach is dilated chiefly in the direction to which the greatest force is applied, downward and laterally. The muscular fibers first elongate in the vertical direction and the distance between the lesser and the greater curvature is increased. Dilatation may also ensue in the transverse and anteroposterior dimensions, and the pylorus may be a little further to the right and in a slightly lower plane, but the lesser curvature maintains its relation to the diaphragm, and this is the differential point between dilatation and gastroptosis (Fig. 181).

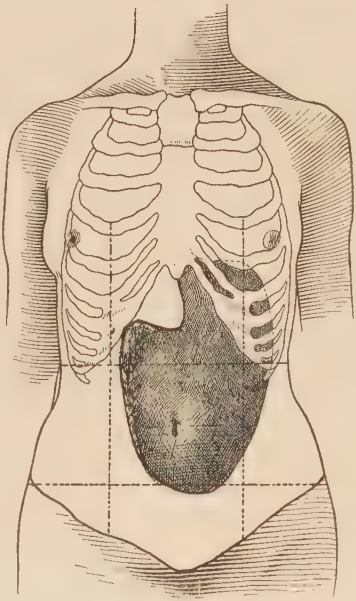

Fig. 181.—Dilatation of the stomach.

There is confusion as to the terms "atony," "ectasy," and "motor insufficiency," as they are often used interchangeably by different authors.

*Atony* of the stomach may be defined, as already stated, as a loss of tone or contractile power of the muscles of the stomach, so that the organ does not contract about its contents, with a resulting *motor insufficiency* (*inability to expel its contents within the normal limit of time*). This condition has not progressed to dilatation.

*Ectasy* may be defined as dilatation, an enlargement of the stomach permanent in character, combined with motor insufficiency. With the atonic type of ectasia, there is motor insufficiency.

*Relative Motor Insufficiency.*—When there is dilatation of the stomach due to obstruction at the pylorus, the motor power of the stomach is not sufficient to expel the stomach-contents within normal time limits. This is spoken of as insufficiency.

This should be considered a relative insufficiency, as in this type the musculature of the stomach is hypertrophied, especially at the pyloric end, and the contractile power is often increased, but not sufficiently to expel the contents past the obstruction within the normal time.

In the dilated stomach without pyloric obstruction we have the true atony of the musculature, with varying degrees of motor insufficiency.

Some claim that stenosis exists in all cases of dilatation of the stomach, but it is easy to demonstrate that there are two distinct types of chronic dilatation of the stomach differing in symptoms and pathologic findings:

(1) The atonic type of chronic dilatation of the stomach. (2) The stenotic (obstructive) type of chronic dilatation of the stomach.

**Differential Diagnosis.**—*Atonic Type.*—In the atonic type there may be few or no symptoms pointing directly to the stomach, the patient frequently suffering from nervous symptoms due to autointoxication and from intestinal disturbances. I have seen many such cases at the Manhattan State Hospital continue a year, or even four or five years, without vomiting. There are no peristaltic waves and cramp-like pains such as occur in the stenotic type followed by vomiting; though some may have dyspeptic symptoms and rarely an attack of vomiting. The postmortem shows the stomach often enormously dilated, with thin walls and no evidence of pyloric stenosis.

*Stenotic Type.*—In the stenotic (obstructive) type of dilatation dyspeptic symptoms are marked, there are peristaltic waves and cramp-like pains preceding vomiting of large quantities of gastric contents, thirst, etc.

In the benign type of long duration the patient often suffers markedly in nutrition, but the cachexia and other symptoms of malignancy are absent and the disease runs a long course. The postmortem shows considerable hypertrophy of the musculature at the pyloric end of the stomach, and elsewhere thinning and dilatation of the muscular wall with evidence of stenosis at the pylorus, or constriction from some external factor.

## ATONIC DILATATION OF THE STOMACH

**Etiology.**—As causes, we may have a primary reduction or loss of muscle power or impairment of it from overwork, for example, from the ingestion of too much material. It directly follows chronic atony of the stomach, which is the preliminary stage, and hence there are similar etiologic factors, such as wasting disease, tuberculosis, chronic gastritis, heart disease, etc.

Among other causes are bolting the food, frequent overloading the stomach, excessive drinking of large quantities of fluid, especially of those containing much gas; it may rarely be congenital; it is quite frequently associated with rickets, in which case gastroptosis is quite often also present. It sometimes follows repeated attacks of acute atony or acute dilatation.

Nothnagel traces back some cases of chronic dilatation to improper methods of feeding during early life. Atonic ectasy we frequently find among the insane. I have found an enormous number of patients at the Manhattan State Hospital suffering from this condition, and quite a number of the women with gastroptosis, and frequently dilatation associated with it. Examinations of many hundreds of cases during the last five years show that very few stomachs were in the normal position or possessed normal functions. Many of these cases have never vomited, and in very many no special symptoms directed attention to the stomach. Unquestionably the *habit of bolting the food common to such patients is a frequent cause of ectasy.* Among the acute melancholics, in whom some ultimate cures resulted, auto-intoxication, *in some cases the result of ectasy with fermentation or putrefaction,* was the primary factor in the production of the nervous symptoms. In the epileptic ward one case suffering from dilatation (atonic) with gastroptosis, and absence of free hydrochloric acid, had suffered from numerous epileptic convulsions both day and night, averaging 140 seizures per month. Under simple diet, initial lavage, and later diet and medication directed to the gastro-intestinal tract alone, she had no convulsions for two years and a half, with the exception of one week, some fourteen months after treatment was begun, during which period she was taken off diet and medication while in the general hospital ward with an acute nephritis. Bromids were only given two months at the commencement in small dosage to break the convulsive habit. At the end of three years the patient was discharged. There were no convulsions, and her mental condition was apparently excellent. I have also another case of epilepsy with atonic ectasia and hypochlorhydria, who has gone over two years without a seizure under gastro-intestinal treatment alone.

Even in the incurable insane this atonic type of dilatation has a direct bearing on some of the symptoms. In a series of 13 paretics, examined for the late Dr. Dent at the Manhattan State Hospital,[1] I found 11 cases of atonic dilatation of the stomach and 2 cases of gastroptosis, and in all secretory derangements of the functions of the stomach; 11 of these cases had at some time of the day a temperature of 99.5° F. and upward, in one 102.5° F. Under treatment directed to the gastro-intestinal tract the temperature was lowered in all 11 (in some to normal), the convulsions, which were present in 5 cases, were diminished in frequency, and in one patient suffering from attacks of syncope, cessation of attacks followed treatment.

The atonic type of ectasy is quite common in many nervous cases, and is undoubtedly, in some, the cause of the auto-intoxication, and the nervous condition is secondary. Atonic ectasy may, therefore, be in many cases the cause of various nervous conditions. On the other hand, ectasia resulting from insufficient mastication and bolting of food among many of the nervous and insane may be a factor in the production of a vicious circle. Beer drinkers and diabetics suffer from this type of dilatation. Professional men, bankers, and brokers, from their irregular habits and rapid eating, are quite liable to this form. The musculature of the pylorus, as we know, is much thicker than other parts of the stomach-wall, and the latter naturally gives way and distends more readily. This does not constitute, however, a stenosis at the pylorus. Ultimately, however, the pylorus itself relaxes in these atonic cases, which undoubtedly accounts for the usual absence of vomiting in these patients.

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[1] Proceedings of the American Psychological Association, Sixty-first Annual Meeting, April, 1905; also the Medical News, July 8, 1905.

This type of ectasy is extremely common.

**Symptoms** are at times not referred to the stomach at all, and often point to the nervous system, or cause an exacerbation of a preëxisting nervous condition. The patient is often neurasthenic or melancholic, with the symptoms associated with these conditions. I have seen the lower border of the stomach in atonic ectasy reach nearly to the symphysis. The following symptoms are generally associated:

Constipation usually marked, rarely diarrhea, coated tongue, frequently headache, and at times dyspeptic disturbances, such as belching and pressure after eating, though often these symptoms are absent.

In a few cases of extreme dilatation there may be occasional vomiting of large quantities of fluid. There are no spasmodic pains, no peristaltic waves, and no marked vomiting, as in the stenotic type. In atonic ectasia with chronic gastritis, the gastric symptoms of the latter may be present, in addition to the other symptoms already noted. More rarely there may be hyperchlorhydria with atonic ectasy. I have found a number of such cases among the epileptics, there being no vomiting, or rarely so, and no peristaltic unrest. They suffer from the symptoms of hyperchlorhydria, motor insufficiency is present, there may be spasmodic pains and vomiting of considerable severity, the vomitus being very acid. These cases, as already noted, have been mistaken for gastrosuccorrhea. After lavage, and later by aspiration of the contents of the empty stomach, one readily demonstrates that hypersecretion is not present. The symptoms in this type are due to the extreme hyperchlorhydria and to *spasm of the pylorus resulting.* Excellent results are secured *by lavage* and by the *treatment of the hyperchlorhydria.* Most of the so-called cures of stenotic ectasia belong, I believe, to this class. The mild types belong to the atonic class, though they might be considered border-line cases. In the milder cases in which cure results without operation, organic changes at the pylorus, I believe, must necessarily have been slight, while the severe cases with peristaltic unrest and progressive symptoms should be classed under stenosis. These last cases which do not rapidly respond to treatment of the hyperchlorhydria, should be referred to the surgeon. Treatment of pyloric spasm hy Einhorn's pyloric dilator the writer believes useless.

**Gastric Contents.**—The gastric findings are variable; fermentation is quite frequently present and hypochlorhydria, or nearly complete absence of free HCl; chronic gastritis, occasionally hyperacidity; mold has been found, and in cases with gastroptosis and dilatation at Ward's Island I have even noted achylia.

The usual manifestations are toxemic in character, with intestinal fermentation or putrefaction and indicanuria.

**Course.**—The milder cases of atonic dilatation are quite amenable to treatment and can be cured. The severe cases do not run so favorable a course, and I have seen such among the nervous and insane where the dilatation extended nearly to the symphysis. I believe that in this type drainage by gastro-enterostomy is indicated, since lavage and diet are only palliative; or in some gastroplication.

In the cases of ectasia due to benign stenosis there is at times, under treatment, a temporary improvement. The stomach, from an increased hypertrophy of the muscles and a subsidence of the hyperemia at the pylorus, may secure a certain amount of compensation and empty its contents fairly well for a time, but usually the symptoms return, and, finally, resection of the pylorus or gastro-enterostomy become imperative in order to save the life of the patient. I have known these cases to drag along twelve to fifteen years without operation. They become chronic invalids. Only by operative procedure can a practical cure of the case be accomplished.

If the stenosis is malignant, then the course depends on the extent of the disease. Even in those cases where removal cannot be undertaken, gastro-enterostomy will relieve the symptoms.

## OBSTRUCTIVE TYPE (STENOTIC) OF ECTASIA

The stenosis causing this type of dilatation of the stomach may be in the gastric tissue at the pylorus or near the pylorus in the duodenum, or it may be external to the stomach—intrinsic and extrinsic causes. The factor causing constriction may be benign or malignant in character, which would modify the clinical symptoms.

**Etiology.**—(1) *Congenital stenosis of the pylorus* (*hypertrophic stenosis*).

(2) *Acquired stenosis*, such as from ulcer, cicatrices following burns from acids or alkalis; from severe gastritis, causing hypertrophy at the pylorus; repeated spasmodic closure of the pylorus due to hyperacidity (spastic stenosis due to inflammation or irritation); benign tumors; pedunculated polypi; adhesions at the pylorus; external tumors; pressure from large gall-stones (in the gall-bladder); perigastric adhesions; spider-web adhesions (Morris) from the gall-bladder; stenotic hypertrophic gastritis (Boas), a fibrous disease of the pylorus (linitis plastica); sclerosis in the pyloric end of stomach (Ottinger); pressure from external tumor; malignant disease of the pylorus.

(3) *Stenosis of the duodenum* from ulcers, cicatrices, carcinoma, external compressions, adhesions, kinks, or diverticula.

It is the modern consensus of opinion that bile may regurgitate into the stomach even if the stenosis is present at the pylorus, as the cicatricial tissue holds it open in many cases; so its presence or absence does not always aid us as to locating whether the stenosis be pyloric or duodenal. Continuous regurgitation of bile and pancreatic juice, however, is suggestive of obstruction of the duodenum below the common duct.

*Movable kidney* I do not believe has any relation to ectasy, but when associated with so-called dilatation, it can be demonstrated that the latter is really a gastroptosis.

**Pathology.**—In the stenotic type, the musculature at the pyloric end of the stomach is much thickened; the fundus is much thinner than normal. The pathologic findings at the pylorus vary according to the cause of the stenosis. The intestines are pushed downward and the liver slightly upward when marked ectasy is present.

**Symptoms.**—The symptoms of dilatation of the stomach due to pyloric stenosis are quire characteristic, but are modified if the condition is cancerous, or in the special type described by R. T. Morris, spider adhesions with hemorrhage.

*Congenital Stenosis of the Pylorus.*—The *symptoms of congenital stenosis* of the pylorus are rather acute in their progressive severity. They usually come on directly after the birth of the infant or a few weeks later, depending on the degree of stenosis. The condition is most readily confounded with a rapidly progressing marasmus. There are present: wasting, the infant rapidly losing weight, projectile vomiting, visible gastric peristaltic waves, non-fecal and no curdled bowel movements, with, *in some cases*, a *palpable tumor mass* in the region of the pylorus. Projectile vomiting occurring early and apparently without cause in an otherwise healthy appearing breast-fed infant, when it can be proved that the mother's milk is without fault, should always excite suspicion of this condition. In cases in which it is impossible to detect the pyloric thickening, even when a whiff of chloroform is administered, and the bowel movements are at times fecal and contain occasional curds, and there is no rapid loss of weight and strength, in spite of the projectile vomiting and peristaltic waves, one may probably assume the condition is due to pylorospasm. These cases, I must say, are difficult to diagnose, and may go on to a slow recovery unrecognized, being considered cases of difficult feeding. As an aid to diagnosis, one may say that, if the infant's weight and strength remain practically the same during medical treatment—*i. e.*, lavage twice daily, the temporary use of the nutritive enema, and later careful mouth-feeding—then the assumption that the condition is due to pylorospasm, either spastic or intermittent, would be correct. Hyperchlorhydria is believed to be a factor in some cases. Dilatation of the pylorus has been suggested by Einhorn. The writer *does not find it practical*, and in the attempt on a young infant one encounters insuperable difficulties. If there *is, however, a progressive loss of weight and strength*, operation should *not be delayed*. Personally, I am very skeptical regarding the curability of *congenital pyloric stenosis* by medical means, and believe the so-called cures to be simply cases of pylorospasm which have recovered. I have no faith in attributing dilatation of the stomach in adults to a congenital stenosis, believing that a careful investigation will show some other cause for the condition.

*Acquired Stenosis of the Pylorus.*—The usual symptoms of *acquired stenosis* are thirst, dryness of the throat, dry skin, oppression, feeling of cramp-like pains of considerable severity, generally associated with peristaltic restlessness of the stomach, eructation of odorous gas, vomiting of considerable chyme, often containing remnants of food taken the day before. This may occur from once to several times a day. The bowels are extremely constipated. Emaciation may become very marked and the loss of fat on the abdomen so great that the skin is in dry wrinkled folds. The urine becomes markedly diminished in advanced cases; its reaction is frequently alkaline.

Intestinal fermentation and putrefaction with indicanuria are often present.

Bradycardia and dyspnea (cardiac asthma) at times are present, as are also stupor, headache, and so-called gastric vertigo.

Tetany or epileptiform attacks may complicate the condition.

The benign type of stenotic dilatation is characterized by rather a long course, with often considerable *temporary improvement under treatment, with a tendency to relapse.*

In the *malignant type* we have the marked cachexia and rapid loss of weight within a few months, the age of patient usually over forty or forty-five, and the character of the vomitus to be noted later.

In my experience the cases characterized by attacks of gastrosuccorrhea are more frequently associated with ulcer at the pylorus with benign stenosis.

*Special Type.*—In an interesting type, to which Robert T. Morris has called attention, there may be vomiting of considerable blood, pain, etc., suggestive of an *active ulcer*. Gall-bladder spider adhesions have been found to be the factor. The symptoms are probably explainable by circulatory interference. The history of previous gall-bladder disease should be examined into.

**Examination of the Gastric Contents in Malignant Pyloric Stenosis.**—Coffee-ground vomit, or the presence of occult blood; free HCl markedly diminished or absent, lactic acid present; few or *no sarcinæ* and the Boas-Oppler bacilli present, in connection with the clinical symptoms, are diagnostic of malignant stenosis. Meat is undigested. Free HCl may be present, especially in the early stages, or when the carcinoma is engrafted on an ulcer.

With benign stenosis we have: the gastric contents separating into three layers, the upper being gaseous; HCl is marked (hyperacidity); yeast and *sarcinæ* are abundant; undigested starch is present. Mold may be found and occasionally bile or sulphuretted hydrogen.

I agree with Einhorn that bile can enter the stomach in some cases of stenosis of the pylorus, the thickened tissue allowing a slight patency.

### DIAGNOSIS

Ectasy means dilatation of the stomach combined with motor insufficiency.

We must, therefore, first determine the position of the organ. Frequent errors have been made in differentiating dilatation and gastroptosis. The position of the upper border of the stomach is the chief point.

There is a simpler method which is dependent on whether or not a movable kidney be present. The movable kidney is almost invariably part of a general ptosis of the viscera (splanchnoptosis), and movable kidney from traumatism is extremely rare.

If we find a movable kidney with a stomach whose lower border is in an abnormal position (too low down), the diagnosis is gastroptosis. If no *movable* kidney is present and the greater curvature is low down, plus motor insufficiency and symptoms, the condition is one of dilatation.

An extremely simple method of locating the lower border of the stomach is by the *splashing sound* (succussion). This has been fully described, and also the creation of the splash for diagnosis. Dehio's method can be used as a check.

We may employ in addition:

(1) *Inspection*, which will in some cases show the outline of the distended stomach, especially after distention with carbonic acid gas. *Active peristalsis is also evident on inspection and is diagnostic of pyloric obstruction.*

(2) *Palpation.*—By this means the peristaltic movements may at times be felt, as can also the cushion-like resistance of the distended stomach. Occasionally a small oval tumor can be determined in benign stenosis, though generally it is not appreciable. With carcinoma of the pylorus the hard resistant mass can often be appreciated.

(3) *Percussion* and *auscultatory percussion*, especially before and after the addition of water, as already described, are useful. The "scratch method" is of service.

(4) *Inflation.*—The stomach may be inflated with air or carbonic acid gas. The outlines can thus be more readily determined and the position of the upper curvature mapped out. Inflation with air or water renders a tumor, if present, more evident to percussion and palpation if it lie on the anterior wall. It disappears if posterior.

## TABLE OF DIFFERENTIAL DIAGNOSIS

| | *Atonic Dilatation.* | *Stenotic Dilatation.* | |
|---|---|---|---|
| | | *Benign stenosis of pylorus.* | *Malignant stenosis of pylorus.* |
| Duration. | Long unless recently acquired. | Quite long, two years, generally considerably more. | Short, few months to one year or one year and a half. |
| Course. | Long, often. | Generally intervals of quiescence or improvement. | Progressive. |
| Tumor. | None. | Occasional (small and smooth). | Present; later palpable. |
| Pain. | Absent. | Spasmodic attacks. | Always present and exacerbations. |
| Peristaltic restlessness. | None. | Present, marked when advanced. | Present and at times marked. |
| Vomiting. | Most frequently absent. | Frequent. | Fairly frequent. |
| Cachexia. | None, but some loss of weight. | None, but great emaciation. | Present. |
| Symptoms. | Often toxemic, referred to nervous system and not specially referred to stomach; at times gastric. | Marked gastric. | Marked. |
| Blood. | None in vomitus. | None except in gall-bladder adhesion cases. | Coffee-grounds in vomit or occult blood.[1] |
| Gastrosuccorrhea. | Rare. | More frequent with ulcer. | Generally absent, though occasional. |

### GASTRIC CONTENTS

| | *Atonic Dilatation.* | *Benign Stenosis.* | *Malignant Stenosis.* |
|---|---|---|---|
| Total acidity. | Lessened; more rarely increased. | Increased. | Generally diminished. |
| Free hydrochloric acid. | Often hypochlorhydria; more rarely hyperchlorhydria. | Generally increased, hyperchlorhydria. | Usually absent. |
| Lactic acid. | At times present. | Absent. | Present, usually marked. |
| Fermentation. | Often marked. | At times marked. | At times, depending on location of growth. |
| Odor. | Often present. | Unpleasant. | Fetid at times. |
| Boas-Oppler bacilli. | Occasional. | Rare. | Usually present. |
| Mucus. | At times, if gastritis. | At times, if gastritis. | In some cases. |
| Sarcinæ. | Present often. | Present markedly. | Usually absent. |
| Yeast. | Marked often. | Often present. | Pronounced yeast fermentation rare. |

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[1] Blood or occult blood present in stool in cancer.

(5) *Transillumination.*—This method, especially with the circumscribing gastrodiaphane and fluorescein, readily determines the outline of the stomach and differentiates between dilatation and gastroptosis in disputed cases. The use of *mensuration by means of stiff sounds is deprecated.* The *x*-rays give no additional information over the methods described, and the expense is an objection.

The determination of the motor functions is most important, *as motor insufficiency is a salient feature.* There are different degrees of this motor insufficiency, and this is best determined by the test-breakfast or test-meal.

*Test-breakfast.*—Ewald's test-breakfast. Aspirate the contents one hour later.

(1) *Normal position of the stomach,* with a residuum aspirated of 100 c.c. or over, and symptoms, show atony of the stomach.

(2) Descent of the lower border of the stomach; 100 to 150 to 200 c.c. residuum or more, with symptoms, and with kidneys in normal position show dilatation of the stomach.

(3) If movable kidney, gastroptosis is present.

Six or seven hours after Leube's test-meal the healthy stomach should be found empty. If undigested food is found (300 to 600 c.c. or more), insufficiency is present and the degree of insufficiency is indicated by the amount of residuum.

It is always preferable to wash the stomach before the test-meal, so as to get rid of the old residuum and make an accurate test. If considerable residuum be found at the end of seven hours, a further test should be made. Wash the stomach and directly thereafter give at 10 P. M. a light supper—a little soup, a slice of bread, a slice of beef, and a little chopped spinach; a small amount of boiled rice or a dozen raisins may be substituted for the spinach. Aspirate and wash the stomach twelve hours later before breakfast. In some cases there will be a marked residuum after seven hours, *but none after twelve hours;* in others there will also be considerable after twelve hours, showing different degrees of insufficiency.

The stomach should be washed, as well as aspirated, to remove all the contents. Also aspirate the empty stomach to test for hypersecretion.

The salol and olive oil tests are not as reliable.

## TREATMENT

The treatment of chronic dilatation of the stomach varies considerably, depending on whether it be due to atony or to benign or malignant stenosis.

**Atonic Dilatation.**—This is by far the most frequent type of dilatation which we are called upon to treat, especially among bankers, brokers, and professional men, who habitually overeat, bolt their food, or are heavy drinkers. Associated with or having a direct bearing on this condition, we may find hypochlorhydria, hyperchlorhydria, or, at times, chronic gastritis. Some of these cases are, in their incipiency, of rather mild type, and prophylaxis, as regards avoiding rapidity of eating and eliminating indigestible food and overeating, is of value. If the patient is run down or anemic, iron and tonic treatment are indicated.

**Diet.**—Though some have recommended a so-called dry diet in dilatation of the stomach, it is a well-known fact that liquids are first evacuated from the stomach, then mushy food, and finally solid food, and this scientific knowledge should be our guide in feeding such cases. Water and food soluble in water leave the stomach soonest of all.

Large quantities of fluid should not be given at a time lest they overdistend the flaccid stomach, but if they are administered in smaller quantities at frequent intervals, a considerable amount can be employed.

It has been demonstrated that alcohol, sugar, and dextrin cause a secretion of water in the stomach.

Milk has been usually recommended as the standard diet in this condition as possessing highly nutritive properties, and the statement has been made that it does not stay in the stomach much longer than plain water.

Penzoldt has demonstrated that water, cocoa, meat broth, soft-boiled eggs, and boiled milk (100 to 200 gm.) leave the healthy stomach within one to two hours, cooking altering the curd formation.

Raw milk takes a considerably longer period, and curds have been found frequently in the normal stomach two or three hours after ingestion.

Experiments have been conducted on my service at the Manhattan State Hospital in cases of dilatation of the stomach, and the periods for the raw milk to remain in the stomach were investigated. After three hours large masses of curd were aspirated.

If the milk were diluted one-half with water, the residuum found at a certain period was just one-half as much as when pure raw milk was used, which formed curds. The higher the dilution, the greater the quantity passed from the dilated stomach within a definite time. Another objection is that 1 liter of milk only represents about 640 calories, and too large an amount would be required if sufficient nutrition is to be obtained from milk alone. Strained soups and strained gruels are evacuated more rapidly. If milk be given, it should, preferably, be combined with some strained gruel or the latter made with milk, so that the nutritive value may be increased.

In the severer type of cases the diet suggested by Seibert in typhoid appeals strongly to the author. It possesses considerable nutritive value, namely:

Strained rice, 8 ounces (250 c.c.), barley or oatmeal soup containing the extract of ½ pound of meat and the yolk of a fresh egg. This can be spiced slightly to improve the flavor. It can be given five or six times daily.

Strained pea soup, lentil, tomato, or potato soup can be used in addition.

Rice flour is excellent in the form of a thin gruel, and can be made with milk which has been thoroughly boiled. The object should be to give frequent (five or six smaller) meals, so as not to overburden the stomach, and yet secure a sufficient amount of nutrition to improve the patient's physical condition.

Cream, 2 ounces (60.0) in 4 ounces (125 c.c.) of water, possesses considerable nutritive value.

Crackers heated thoroughly and well buttered can be rubbed up in the broth.

Fat in the form of cream and butter should be administered.

In the *milder cases*, scraped beef, rare beef, soft-boiled eggs thickened with *a small amount of mashed potatoes*, and rice strained through a colander, with plenty of butter, can be given, with a little asparagus and spinach. Other vegetables are more difficult to expel from the stomach.

Matzoon, koumiss, bacillac, kefir, and milk prepared with lactone tablets (lactone-buttermilk) are of special value in cases suffering from auto-intoxication, having nervous symptoms and indicanuria (intestinal putrefaction). The matzoon can be diluted with one-third water or Vichy that has been allowed to become flat. This last avoids gaseous distention of the atonic stomach. It is also preferable to allow some of the gas to pass off from the koumiss. About 1 quart of one of these preparations can be used daily—additional water or Vichy one-third in volume being then added. These sour milk preparations, especially with the slight dilution, pass readily from the stomach. They do not curdle like plain milk. The yolks of several raw eggs, stale bread or crackers with plenty of butter and cream, strained vegetable soups, rice gruel, and sanatogen can be added. Meat preparations should be avoided in these cases.

In cases with deficiency of hydrochloric acid, the meats are not well digested and should be given in smaller quantities; rice, barley, and tapioca (strained) or in purées, and mashed potatoes are of service, and in larger amounts. I have often found raw eggs beaten up in water or milk of great service, employing at times six to eight daily. The milk can be completely or partially peptonized to lessen curd formation. With the precaution noted, milk may be employed in the cases with not too marked motor insufficiency. It is a simple matter to test whether it leaves the stomach readily or not.

If thirst is marked, rectal enemata of hot normal saline solution are indicated, and in very severe cases the stomach may be given a rest and nutritive enemata be given for a few days. Proctoclysis is of value for the thirst.

Sanatogen, preferably flavored, tropon, and somatose are useful adjuncts, given in divided doses in the broths. If anemia is present, iron tropon can be added, 1 dram (4.0), three times a day. Always peptonize the milk if given by enema.

After eating, the patient should lie down for from one-half an hour to an hour, preferably on the right side, so the stomach can empty itself more readily.

**Mechanic Support.**—One of the most important methods of treatment is the use of proper support to the organ, and the ideal method is by Rose's adhesive plaster belt, a description of which has been given. It *increases intra-abdominal pressure* and the stomach is pushed upward, acting in effect *like gastroplication.* Transillumination was employed in one case (Fig. 182), the belt applied, and transillumination again carried out (Fig. 183).

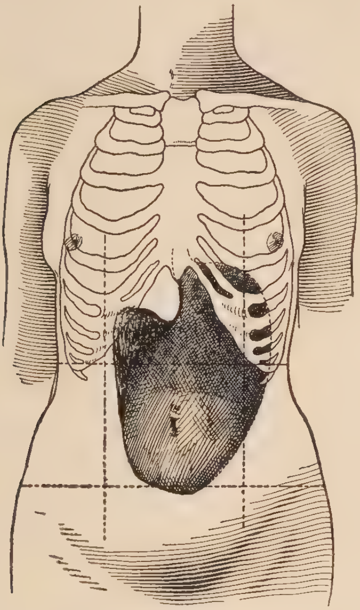

Fig. 182.—Dilatation of the stomach. Transillumination with fluorescein before application of Rose's belt (Case 1) (Ward's Island Gastro-intestinal Clinic, Manhattan State Hospital).

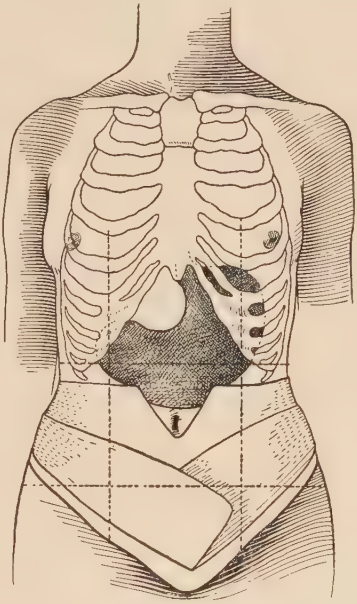

Fig. 183.—Dilatation of the stomach, same patient (Case 1). Transillumination with fluorescein after application of Rose's belt. By accurate measurement the stomach has been elevated and the lower border is 4 inches higher than it was before the belt was applied. The lower border now lies above the umbilicus (Ward's Island Gastro-intestinal Clinic, Manhattan State Hospital).

The illustration (Fig. 183) shows the result, the lower border of the stomach being elevated 4 inches. Though Pancoast, Sailer, and Worden[1] claim that the $x$-ray has determined that no form of belt, binder, or corset elevates the stomach to the smallest extent, in the experiments described after illumination of the stomach without support, the patient was then *placed in the Trendelenburg position,* and Rose's belt applied. It was clearly demonstratd that elevation had then occurred. This belt should be worn four or five weeks and a new one then applied. It aids in the evacuation of the stomach-contents. Silk abdominal belts may be substituted, but the support is not continuous and the adhesive strapping is superior. From the *positive results* secured by the writer, he can only assume that if adhesive strapping was employed, the technic was not correct in Pancoast's cases. Undoubtedly many of the belts and corsets are useless.

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[1] Trans. Coll. Phys., Philadelphia, 1906, series 3, xxviii, 151–196.

**General Hydrotherapy.**—External douches—the fan douche and also the Scotch douche applied to the region of the stomach and changing the temperature of the water—have been serviceable in some cases. Cold compresses and cold sponging are at times useful. An adjustable silk belt is worn in such events.

**Local Treatment of the Stomach.**—(1) *Lavage.*—In some of the milder atonic cases proper diet, mechanical support, and appropriate medication may suffice without or with occasional lavage. In the more severe cases lavage is indicated, and the time of its performance and frequency depend upon the degree of dilatation and the amount of residuum found after the test-meal (degree of motor insufficiency). If there is not immediate improvement if lavage is omitted, it must be added to the treatment.

If a large amount of residuum is present after a test-meal or test-breakfast, or there are nervous symptoms, or in the morning before breakfast a residuum is present, lavage should be carried out.

As to the proper hour for lavage, I believe Riegel holds sound views, and my own experience agrees with his; if the residuum is 200 to 500 c.c. or more before supper, it is best to wash the stomach then and follow with a light meal; if this is not done, the organ will contain fermenting food during the night which will increase the atony. If food is present before breakfast a second lavage is then indicated.

The washing should be performed with the patient both sitting and lying down, so as to remove all the irritating material. The stomach should be washed until the return is clear.

Daily lavage is generally necessary at first. As the tone of the stomach improves, the residuum found will decrease and washing may be performed less often. The first part of the washing may be done with plain warm water, though normal saline solution is preferable.

I have found milk of magnesia (Phillips), 2 ounces (60.0) to 1 quart (liter) of water, excellent for the preliminary lavage. It is well to employ antifermentatives in the final treatment, such as—

| | |
|---|---|
| Acid salicylic | 1 : 1000 |
| Sodium salicylate | 1 : 1000 |
| Sodium benzoate | 1 : 1000 |
| Listerin | ʒj (4.0) to 1 quart (liter). |
| Glycothymolin | |
| Borolyptol | |
| Resorcin or | gr. 15 to 30 (1.0–2.0) to 1 quart (liter). |
| Boric acid | |

(2) *Electricity.*—The intragastric faradic current (preferably) can be employed if there be no objection on the part of the patient, using Lockwood's instrument. In many cases the percutaneous method is advisable.

*Static electricity* is claimed to be of value, and in some cases to reduce the size of the atonic dilated stomach.

*Massage* or *vibratory massage* is of service to tone the musculature and aid in emptying the stomach.

(3) The *stomach douche* has been recommended in the milder forms to stimulate the organ. The fluid should be at a temperature of 95° to 85° F. (gradually reduced).

If hydrochloric acid is diminished, normal salt solution may be employed; if HCl is increased, then use silver nitrate 1:3000 to 1:2000.

Bitter remedies, such as quassia (fluidextract), 15 minims to ½ dram (0.888–1.77 c.c.), or a cup of quassia-water (quassia cup filled with water and allowed to set for half an hour); or hops (fluidextract lupulin), 15 minims to ½ dram (0.888–1.77 c.c.), or fluidextract of condurango, ½ to 1 dram (1.77–3.54 c.c.) to a liter of water, have been recommended for lavage as a stimulant, but I see no special value in their use.

The *stomach spray* has also been suggested in place of the stomach douche, but it possesses the disadvantage of injecting considerable air.

*Medicines.*—If there is deficiency of hydrochloric acid, the stomachics and hydrochloric acid should be administered, such as are employed in chronic gastritis. The following prescription is often valuable:

℞. Tinct. nucis vomicæ }
Acid hydrochloric, dilute } ............... āā 12.0 (ʒiij);
Comp. tinct. cinchona ..................... 16.0 (℥ss);
Aq. destil ........................ q. s. ad. 125.0 (℥iv).—M.

Sig.—One to two teaspoonfuls in a wineglassful of water t. i. d. half an hour before meals.

Oxyntin with nux vomica, one to two capsules t. i. d., before or after meals, is useful.

If there is hyperacidity, magnesia usta, 15 grains to ½ dram (1.0–2.0) or more, in water t. i. d. an hour after meals. Or milk of magnesia (Phillips), 1 to 2 drams (4.0–8.0), in water. These are excellent remedies.

If bicarbonate of soda be employed, it is better to combine it with magnesia usta, equal parts, as it readily generates carbonic acid gas.

In the *atonic type* of dilatation I have always been inclined to employ nux vomica or its alkaloid as a stimulant to the musculature of the stomach, even though hyperacidity be present, which is rare;

in the latter event combining belladonna with the nux, as a pill, before meals:

℞. Ext. nucis vomicæ } ..................... āā gr. $\frac{1}{4}$ (0.016).—M.
Ext. belladonna }

or

℞. Strychnin ................................ gr. $\frac{1}{60}$ (0.00108);
Atropin ................................ gr. $\frac{1}{100}$ (0.00064).—M.

If nux vomica is incorporated in the stomachic mixture, it should not be repeated. If the tincture of nux vomica is employed, it can be gradually increased to large dose—$\frac{1}{2}$ dram (2.0) t. i. d.

For *fermentation* and intestinal putrefaction the following remedies are of service, given three times a day half an hour to an hour after meals: Salicylate of soda, benzoate of soda, ichthoform, ichthalbin, bismuth salicylate, salol, resorcin, benzonaphthol, bismuth phenolate, or bismuth sulphocarbolate, all in doses of 5 grains (0.3) each. Urotropin, 5 grains (0.3), given in combination with sodium benzoate, 5 grains (0.3), in water t. i. d. after meals, is also useful.

I have found resorcin an excellent remedy, alone or combined with bismuth subnitrate. If mold is present the creosote preparations, as suggested by A. Rose, are preferable, such as beechwood creosote, 1 minim (0.059 c.c.), or carbonate of creosote (creosotal), 5 grains (0.3), three times a day after meals.

For *constipation* the olive oil injections at night, to be retained, 4 ounces to 1 pint (125–500 c.c.) or more, as suggested by Fleiner; massage, electricity, the establishment of a regular hour for stool, the administration of a glass of water on rising, and, if required, the use of the cascara preparations or the aloin and belladonna pill, regulin, or one of the phenolphthalein preparations, such as phenolax or purgen, at night. The saline cathartics are objectionable.

Gastrosuccorrhea is rare in the atonic type of dilatation. In the event of its presence, lavage with nitrate of silver (1 : 2000) twice a week, and belladonna tincture, 10 minims (0.592 c.c.) t. i. d., or extract of belladonna, $\frac{1}{3}$ grain (0.022) t. i. d., are indicated. For further treatment, the chapter on this subject should be consulted.

For gastric tetany, which may occur in the atonic type of ectasy, but which is a rare condition, lavage is of service, but gastro-enterostomy is indicated. Moynihan has operated on 14 cases of gastric tetany, with cures in all.

*Surgery.*—In atonic ectasy, when no improvement occurs under treatment or when the dilatation is of great degree and the patient's condition seems to be getting worse, operation is indicated—preferably drainage of the stomach by gastro-enterostomy.

Gastroplication—infolding the wall of the stomach and sewing it in pleats—has also been successfully reported. Coffey has sutured the greater omentum to the abdominal wall and thus supported the stomach in a hammock. He reports two favorable results.

### TREATMENT OF STENOTIC DILATATION (BENIGN STENOSIS)

The author wishes to preface this section with the statement that he considers all cases of *pyloric stenosis to belong to the surgeon.* The medical treatment is only palliative; though some cases may live many years, especially the milder types, but always be under the care of a physician for a considerable period during each year. In addition, some cases when untreated may become so reduced as to be unfit for operation. Medical treatment, lavage, often twice daily, accompanied by proper methods of feeding by mouth, together with nutritive enemata, may so improve the physical condition of the patient that he can in a short time undergo operation. This was particularly demonstrated in a case of carcinomatous stenosis in which the methods of feeding are described under Carcinoma of the Stomach.

*Treatment of Congenital Stenosis of the Pylorus.*—This is surgical—gastro-enterostomy as early as the diagnosis can be made. In the cases which are more intermittent in character, and which, under medical treatment, retain their weight and strength—the condition may be believed to be due to pylorospasmus. The medical treatment consists of lavage twice daily with a warm weak solution of soda bicarbonate, 1 to 2 drams (4.0–8.0) to the pint (500 c.c.), proper feeding by mouth, nutritive enemata, and small doses of strontium bromid, 3 to 5 grains (0.2–0.3), by mouth or rectum. Correct hyperchlorhydria if present. This method can be tried for a short time in any event, and if the symptoms continue, and the loss of weight and strength are progressive, one may assume the case to be due to stenosis, in which event operation is indicated. Einhorn[1] advocates the use of an infantile pyloric dilating catheter, but the writer does not advocate his method.

*Treatment of Acquired Stenosis of the Pylorus.*—In the cases of acquired stenosis where there is a mechanical obstruction to the exit of the gastric contents, the muscular action is increased during the earlier stages and hypertrophy of the pyloric end of the stomach is present; later the fundus and body become distended and thinner. Gentle massage from left to right, or vibratory massage by the same method, the patient lying on the right side and the manipulations being performed an hour or two after meals, may aid in emptying of the stomach. Electricity is of slight or no value. I have seen no permanent benefit from these methods in the obstructive type of dilatation. Olive oil, 2 to 4 ounces (60.0–125.0 c.c.), administered three or four times a day before meals will aid the passage of the food through the stenosed region, and Rose's adhesive plaster belt is also of service to elevate the stomach.

The diet must be liquid in the worst cases and mushes may be employed in the less severe types. Raw eggs, six to eight a day, plenty of fat, such as butter and cream, sanatogen, somatose, and tropon, are all of service. The general method of feeding with small frequent meals is the same as in atonic ectasy. Improvement in weight must be secured. Solid food is objectionable. "The sour milks, such as bacillac, lactone-buttermilk, kefir, matzoon, and koumiss, administered after the manner described in Atonic Ectasy, are of value." The patient should lie on the right side for about half an hour after each feeding in order to facilitate the exit of the food from the stomach. Lavage is always necessary, and for the attacks of spasmodic pain is the most rapid method to secure relief. As hyperacidity is present, alkalis are indicated to correct this condition, as in hyperchlorhydria.

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[1] Med. Rec., June 10, 1911.

Tincture of belladonna in large doses, 10 to 15 drops t. i. d., will often relieve spasm and pain, as will also the application of heat.

**Vomiting.**—For vomiting lavage is indicated, followed for several days by rectal feeding, and then commencing with a small amount of liquid nourishment.

Rectal injections of normal saline solution, 8 ounces to 1 pint (250–500 c.c.), may be indicated to relieve thirst and collapse. Proctoclysis is of service.

Thiosinamin, 5-grain (0.3) doses t. i. d. by mouth, or 3-grain (0.2) doses in 15 per cent. alcoholic or 10 per cent. glycerinated solution by hypodermic, may prove of service in fibrous contractures of the pylorus. Fibrolysin (thiosinamin and sodium salicylate, Merck), each ampoule containing 0.2 gram thiosinamin, can be given daily or every other day, for a month or two, in cases refusing operation.

Gastrosuccorrhea may occur in cases when there is an ulcer with stenosis at the pylorus and, rarely, hemorrhages. Temporary treatment as for hemorrhage of gastric ulcer is indicated, and then surgical procedure. The treatment for gastrosuccorrhea is described in the chapter on that subject.

Gastric tetany may be a complication for which lavage is indicated temporarily, and then operation (gastro-enterostomy).

I have seen cases of ectasy from benign stenosis lose 75 pounds in weight and regain 50 to 60 pounds under treatment, and ultimately relapse and come to operation. I have treated many patients of this type, so-called brilliant cures by specialists, the ultimate results being the same. They, even at the best, tend to become chronic invalids and always require treatment. *The best physician for these cases is the surgeon.*

If the cause of the stenosis lies external to the pylorus, bands, adhesions, etc., can be separated. If it is intrinsic, resection of the pylorus, pyloroplasty, or gastroduodenoplasty can be performed in suitable cases, or drainage by gastro-enterostomy. The latter is usually the operation of selection. Divulsion I do not approve.

I have seen a patient gain 100 pounds in weight in eight weeks after gastro-enterostomy, and, from being a confirmed invalid, restored to perfect health. Einhorn[1] advocates the use of his dilating catheter or pyloric dilator with diaphane to stretch the stenosed pylorus. The writer knows the method to be uncertain, and the results not permanent. Valuable time is thus lost.

### MALIGNANT STENOSIS

In these cases early radical operation is indicated, as described under Cancer of the Stomach; otherwise, palliative gastro-enterostomy. If operative procedure be refused, then diet, lavage, and the treatment laid down under Carcinoma Ventriculi.

## COMPLICATIONS OF CHRONIC ECTASY

### GASTRIC TETANY

Tetany is characterized by peculiar bilateral tonic spasm of the extremities, either paroxysmal or continued.

**Pathology.**—In all cases there is dilatation of the stomach of a high degree, due generally to stenosis of the pylorus or the duodenum; frequently the result of an ulcer; rarely from carcinoma.

Gastrosuccorrhea has been associated with it in some cases. Tetany has also been reported with acute ectasy and in atonic ectasia.

**Etiology.**—There are three theories as to its cause:

(1) Kussmaul and, later, Fleiner believed its symptoms are due to the great loss of fluid in the system, the thickening of the blood, and the consequent drying of the tissues.

(2) Friederick Müller and Germain Sée consider it to be the result of some reflex action, as Müller brought on an attack by tapping the epigastric region. Riegel has observed it on passing the stomach-tube; and it has also occurred in cases of intestinal worms.

(3) The third theory explains it on the ground of auto-intoxication, since fermentation and putrefaction are present in the stomach.

This last is probably correct, as the cases have been benefited by lavage and cured by stomach drainage (gastro-enterostomy).

Amato reports a case of gastric tetany with death. He has introduced fermenting materials into the stomachs of animals and produced dyspnea, myosis, muscular contraction, and trismus. The liver and pancreas (postmortem) showed lesions, such as are usually found with poisonings and intoxications.

**Symptoms.**—There are tonic and clonic bilateral spasms, which appear suddenly and are generally confined to the extremities, the flexor muscles being chiefly affected. The fingers are bent at the metacarpophalangeal joint, extended at the terminal joints, being pressed close together, and the thumb is contracted into the palm of the hand. The wrists are flexed, the elbows bent, and frequently the arms are folded over the chest. The knees are bent, the feet extended, and the toes adducted.

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[1] Med. Rec., Oct. 9, 1909; Ibid., June 10, 1911.

In severe cases there may be trismus, and the angles of the mouth are drawn up. There is sometimes edema of the hands and feet.

The spasms are usually paroxysmal and last for a variable time. The eyes may be turned up.

In the acute attack there may be a rise of temperature and elevation of the pulse. In some cases there may be involvement of the muscles of the back and of the thorax, with dyspnea and cyanosis.

The attack may be acute, from a few minutes to several hours, but there may be some stiffness and contraction lasting several weeks.

**Diagnosis.**—There are certain diagnostic features:

(1) "*Trousseau's Symptom.*"—As long as the attack is not over, the paroxysms may be produced by compressing the affected parts, either in the direction of their principal nerve-trunk or over their blood-vessels, so as to impede the venous or arterial circulation.

(2) "*Chvostek's Symptom.*"—There is an increase in the mechanical excitability of the motor nerves. A slight tap over the facial nerve will throw the muscles to which it is distributed into active contraction.

(3) "*Erb's Sign.*"—The electric irritability of the motor nerves, to the galvanic current especially, is increased.

(4) "*Hoffmann's Sign.*"—Heightened excitability of the sensory nerves. The slightest pressure may cause paresthesia in the region of distribution.

The **prognosis** of tetany is extremely bad.

**Frequency.**—Moynihan believes it to be not so very rare, and reports 14 cases in which gastro-enterostomy was performed, with a cure in each case; though some claim only 30 to 40 cases are reported.

Tetany-like attacks with epileptiform attacks are more frequent, and will be referred to under Epilepsy.

**Treatment.**—Bromids, and even chloroform inhalation, during the acute attack are of value. Lavage is beneficial.

The chief indication is drainage of the stomach by gastro-enterostomy.

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## CONVULSIONS—EPILEPSY

*Epileptic seizures* occur both in cases of chronic ectasy and in other affections of the stomach. I have had a case under observation at the Manhattan State Hospital who suffered from repeated attacks of epilepsy, 140 seizures a month. There was dilatation of the stomach, with ptosis and hypochlorhydria. For the last three years, while having occasional lavage, dietetic treatment with medication directed to the stomach, no bromids, she has had no attacks, except for the period of a week, over two years ago, when the treatment was omitted. The mental condition cleared up, and the patient was recently discharged. At the same institution there is another patient with atonic ectasia and hypochlorhydria, who had frequent epileptic attacks and who has had no seizures for over two years, as a result of treatment directed to the gastro-intestinal tract.

I have reported a case of hyperchlorhydria,[1] with epileptic seizures, apparently cured by appropriate treatment; and cases of dementia paralytica with chronic ectasy, in which the convulsions were diminished under treatment and the high temperatures returned to normal. I have also seen *tetany-like convulsions* in a case of dementia præcox, with gastroptosis and marked ectasy, improve after treatment of the stomach. Salt-free diet, the avoidance of meat, open air, and treatment appropriate to the gastric findings in each case are indicated.

In conclusion, I cannot recommend too highly the sour-milk diet in the treatment of these cases. Matzoon, koumiss, kefir, or the lactone-buttermilk can be employed after the manner indicated under Atonic Ectasy. I have found Wm. H. Thomson's formula for the preparation of matzoon of great value in these cases. It is as follows:

" 1. Place half a cake of yeast in 1 pint (500 c.c.) of fresh milk in a pitcher covered with a towel in a warm place for twelve hours; then

" 2. Add to this 1 quart (liter) of milk and keep in a warm place for twelve hours; then

" 3. Take 1 pint (500 c.c.) of No. 2, add to it 1 quart (liter) of milk, and keep in a warm place for twelve hours; then

" 4. Take 1 pint (500 c.c.) of No. 3, add to it 1 quart (liter) of milk, and keep in a warm place for twelve hours.

" This makes 1½ quarts (1500 c.c.) of matzoon, the entire process occupying forty-eight hours.

" One quart (liter) of this can be administered in divided doses daily, breaking in it stale bread or crackers, and eating it with a spoon. The remaining pint (500 c.c.), with the addition of 1 quart (liter) of milk at the end of twelve hours, will furnish 1½ quarts (1500 c.c.) of matzoon. Fresh matzoon can be made daily from the former pint (500 c.c.) of mother matzoon for about two weeks, when the process must be started over again. If larger quantities are to be used, 1 quart (liter) of No. 2 can be used with 2 quarts (liters) of milk, and so on."

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[1] Observations on the Relation of the Gastro-intestinal Tract to Nervous and Mental Diseases, reported April 17–21, 1905, American Medicopsychological Association, and in their proceedings.

This method has been extensively employed by Thomson and the author in feeding our epileptics. One can also use 4 to 6 ounces (125–185 c.c.) of the ordinary bottled matzoon to 1 quart (liter) of milk, which will produce matzoon if kept twelve hours in a warm place. From this daily matzoon can be made from each previous supply for about a week, when a fresh bottle must be employed.

## CHAPTER XVI

# ANOMALIES IN THE POSITION AND FORM OF THE STOMACH—HOUR-GLASS STOMACH—DISLOCATIONS—GASTROPTOSIS

ANOMALIES of form are frequently congenital. Fore-stomach is a dilatation of the lower end of the esophagus immediately above the diaphragm. Antrum cardiacum is a sacculated diverticulum of the esophagus situated below the diaphragm. In some cases no symptoms occur; in others food becomes lodged and causes serious results. Megalogastria is a congenital or acquired large stomach with normal functions. Microgastria is an abnormally small stomach with normal functions. Angustatio ventriculi is an extremely small stomach due to stricture of the cardia or esophagus or to cirrhosis ventriculi.

**Congenital Narrowing of the Pylorus.**—This condition is due to hypertrophy of the circular muscles of the pylorus. It may be of so severe a type that ingestion of food may be impossible and the infant die within a few days after birth. In milder cases chronic ectasy may result according to some.

### HOUR-GLASS STOMACH

This condition may be *congenital*, but is more frequently acquired. The stomach has a peculiar sacculated outline, and is divided into two parts—the cardiac and pyloric (Fig. 184). In some cases the cardiac sac is larger, in others, the pyloric.

**Etiology.**—Cicatrized tissue contraction following an ulcer; less frequently peritonitic adhesions, gastritis from corrosive poisons, and carcinoma are causes. Slight forms may produce no characteristic symptoms. In advanced cases the division may be recognized. By the ingestion of bismuth and the use of the fluoroscope with the *x*-rays this condition can be demonstrated. It is advised to secure a röntgenograph when possible.

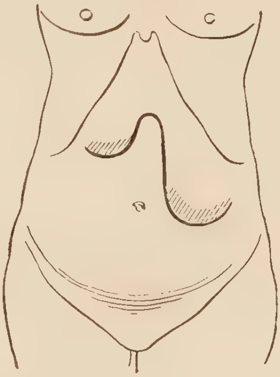

Fig. 184.—Hour-glass stomach.

The following diagnostic points have been given by Moynihan:

With lavage, part of the fluid is lost; if the stomach is washed clean, a sudden reappearance of the stomach-contents takes place, "paradoxical dilatation"; when the stomach has been apparently emptied, a splashing sound may be elicited by palpation of the pyloric segment; after distending the stomach, a change in the position of the distention tumor may be seen in some cases. Gushing, bubbling, or sizzling sounds are heard on dilatation with carbonic acid gas at a point distinct from the pylorus. In some cases, when both parts are dilated, two tumors with a notch or sulcus between are apparent to sight and touch. On both sides of the furrow there will be a loud tympanitic sound which cannot be elicited in the middle.

Stockton finds that if the first part of the stomach is aspirated, after manipulation, it is sometimes possible to force from the second into the first portion a gastric juice of different quality. Operative procedure is the only method of cure.

## DISLOCATION OF THE STOMACH

The fundus may be dislocated upward. Among the causes are:

Absorption of a pleuritic exudate on the left side; after contraction of the lung or any process which is accompanied by upward dislocation of the diaphragm; excessive distention of the abdominal cavity, forcing the diaphragm upward, such as from pregnancy, ascites, tumors, and meteorism; diaphragmatic hernia.

The cardiac end of the esophagus may become bent. Lateral dislocation is rare, and may be caused by tumors of the spleen, distended colic flexure, or lateral pressure from an enlarged liver. As a rule, the latter forces the stomach downward.

**Downward dislocation** (gastroptosis) is the common form.

## GASTROPTOSIS—ENTEROPTOSIS—GLÉNARD'S DISEASE

(*Synonyms.*—Gastroptosis—Rose; Visceroptosis; Splanchnoptosis; Abdominal Relaxation or Atonia Gastrica—Rose; Atony of the Third Degree.)

**Definition.**—Gastroptosis may be defined as a prolapse or downward displacement of the stomach, right kidney or both kidneys, and other organs of the abdominal cavity, which may be associated with disturbances of the gastro-intestinal tract and pelvic organs, together with various nervous symptoms. Ptosis of the heart may also occur.

*Nephroptosis is a stigma of gastroptosis.*

**Introduction.**—I here use the term "gastroptosis" with the usual definition (ptosis of the stomach), though Rose has shown it correctly means descent of the belly (splanchnoptosis). The reader must remember that gastroptosis is a quite frequent condition, and that it may be accidentally discovered in some cases which have no symptoms whatever. On the other hand, there are various degrees of ptosis of the stomach, in some of which the symptoms are rather mild in character, while in others there may be the symptoms-complex of Glénard's disease.

It is not the position of the lower border of the stomach which constitutes a ptosis, but that of the upper border; with the relaxation of the suspensory ligaments of the stomach the lesser curvature sinks as well as the greater, and we may have varying degrees of ptosis, from moderate obliquity of the upper border to a vertical stomach; while on the other hand, the entire organ may sink and give the crescentic form of gastroptosis. The determination of the lower border alone is not diagnostic, since it may merely be evidence of a dilated stomach. Hundreds or even thousands of cases of nephroptosis have been reported as having dilated stomachs, the dilatation being imputed to pressure of the kidney on the duodenum, and no investigation has been made of the position of the lesser curvature.

From my own experience, I do not hesitate to say that movable kidney (nephroptosis), with the lower border of the stomach lower than normal, is diagnostic of gastroptosis. Dilatation of the stomach is often associated with gastroptosis, but kidney pressure on the duodenum, in my opinion, has no bearing as to its production. This combination has been found in cases at the Manhattan State Hospital. Furthermore, treatment for gastroptosis will generally cure this condition. Ptosis of the stomach in some of these cases may be of extremely mild type.

**Anatomic Considerations.**—It is necessary to briefly allude to certain anatomic features. The liver, as we know, is suspended from the diaphragm by ligaments derived from the peritoneum. The cardiac end of the stomach is held quite fixedly in position by the esophagus, and there is a peritoneal attachment to the diaphragm at this point, the gastrophrenic ligament. In this location the stomach lies in close relation to the diaphragm, while the lesser curvature is suspended from the liver by the lesser omentum (gastrohepatic). The spleen lies in close relation to the diaphragm, being attached thereto by ligaments (processes of the peritoneum), and to the stomach by the gastrosplenic omentum.

It is thus readily understood how compression of the lower part of the thorax or effusions above the diaphragm may mechanically force down the latter and produce ptosis of organs so closely associated. The descent of the intestines is a natural accompaniment.

A tumor of the pylorus may cause ptosis of the stomach, and prolapse of the transverse colon and of the other viscera follow.

On the other hand, a severe type of dilatation of the stomach may be followed by ptosis of the organ and then general visceroptosis. These primary types of gastroptosis are not so very frequent.

The transverse mesocolon surrounds the transverse colon and connects it with the back of the abdomen at the spine. The transverse colon is attached to the abdominal surface of the eleventh rib on each side by a fold of peritoneum. As the colon passes across the abdomen it sags somewhat, presenting a slightly concave surface superiorly. Glénard, whom we must justly credit as the first to describe splanchnoptosis as a pathologic entity, believes enteroptosis (ptosis of the transverse colon) to be the starting-point. He thinks the transverse colon is fastened to the pyloric end of the stomach by a band (ligament), and that the hepatic flexure first sags, followed by the transverse colon, causing thus a sharp flexion at the attachment of the ligament, and a hindrance to the progress of the intestinal contents, with resulting accumulation in the ascending and transverse colon. From the point of stenosis the transverse colon passes downward diagonally across the abdomen as a hard cord-like mass (corde colique transverse).

The sagging of the transverse colon exercises traction on the pylorus and omentum, thus causing descent of the stomach and liver. The descent of the hepatic flexure, he believes, causes traction on the parietal peritoneum and encourages ptosis of the right kidney. The gastro-intestinal tract, he noted, was suspended in the form of loops, six in number, by means of ligaments; and he believed in the possibility of too great a bend at such an acute angle, that it might cause a partial obstruction to the passage of the contents. This might occur at the gastroduodenal, the duodenojejunal, transverse colon, or sigmoidorectal curves.

The gastroduodenal and transverse colon ligaments Glénard holds to be the weakest, and if they give way, with resulting ptosis of the intestine, increased traction and angulation is produced at the next fixation point, causing an enterostenosis.

Glénard found the transverse colon displaced and stenosed in numerous autopsies, and was the first to realize that many cases of so-called nervous dyspepsia were dependent upon these abnormalities.

Riegel has demonstrated that the hepatic flexure is *not dislocated* downward in the majority of cases, and Glénard's explanation I hardly believe tenable, as there are other very important factors which have a bearing. A tumor, however, of the transverse colon or adhesions may produce primary enteroptosis.

We must remember there is *one type of case*, a congenital constitutional defect, the patient with long narrow thorax, who suffers from splanchnoptosis.

Keene finds in autopsies on babies evidences in favor of the congenital origin of enteroptosis. In many of these there was a redundant colon. A potential enteroptosis is, therefore, present, which is latent. Later, through some cause, weakening of the abdominal musculature or diminution of intra-abdominal pressure, sagging of the intestine occurs and symptoms develop. The writer believes these congenital cases are undoubtedly to be found. The round-shouldered, hollow-backed position, Reynolds[1] holds, results in the formation of pot belly and leads to the production of splanchnoptosis. Undoubtedly, spinal curvature or rickets may be factors.

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[1] Jour. Amer. Med. Assoc., Dec. 3, 1910.

The major number of cases of gastroptosis, however, are acquired from various causes; and in my opinion the development of the prolapse of the various organs generally occurs synchronously, the stomach, right kidney, and transverse colon most frequently prolapsing together; while in other cases the left kidney or the rest of the viscera may descend in addition.

These following are the prominent factors which have a marked bearing in preserving the proper position of the viscera:

(1) *The abdominal muscles.*

(2) *The maintenance of normal intra-abdominal pressure.*

(1) *Abdominal Muscles.*—In an interesting article, A. Rose[1] calls attention to the fact that in addition to the usual functions described in the text-books, in assisting expulsion of the fetus, bowel action, urination, and vomiting, the abdominal muscles aid in the preservation of the physiologic position of the abdominal organs. The crosswise arrangement of the external and internal oblique and transversalis muscles—supported by the recti—effect a narrowing of the abdominal cavity and prevent visceral ptosis.

Groddeck, of Baden-Baden, has, moreover, described the mechanical influence of healthy muscle, by the alternate contraction and expansion, in assisting the circulation of the blood and lymph, and an atonic condition of the abdominal musculature would certainly interfere with the maintenance of the normal relations between the extra- and intra-abdominal circulation. Moreover, clinically, simple inspection will differentiate between normal conditions and the typic "pot-belly" of the gastroptosis patient. Acute or wasting disease may also cause changes in the muscles.

(2) *Intra-abdominal Pressure.*—Normal abdominal muscles also maintain the normal intra-abdominal pressure necessary to preserve the position of the viscera.

Walkow[2] has made a very exhaustive study of this question and has demonstrated, *for example, on the cadaver,* with the upper part of the trunk elevated (the reversed Trendelenburg position), that after abdominal section, mobility of varying degrees of the kidney is found, which did not previously exist.

Stürmdorf has found similar results after laparotomy on the living.

Clinically, changes in the intra-abdominal pressure, the result of childbirth or tapping for ascites, have resulted in the production of splanchnoptosis, the thinned and distended musculature of the abdomen also being a factor.

Rapid loss of weight from emaciation and absorption of omental fat is another example.

**Nephroptosis.**—Movable kidney, in probably 95 per cent. of cases in my own experience, is one of the stigmata of gastroptosis.

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[1] Surgery, Gynecology, and Obstetrics, November, 1906, Physiology and Pathology of the Abdominal Muscles.

[2] Med. Rec., Jan. 13, 1906.

The congenital type, with long mesonephron, or those cases due to traumatism, are comparatively few in number.

The right kidney has a longer pedicle and lies lower on account of the liver.

Stürmdorf refers to certain skeletal deformities as influencing the shape of the bony receptacle for the kidneys, and which in some cases predisposes to prolapse; but gastroptosis is associated with these same conditions.

It has been claimed that there is a nephrocolic ligament connecting the kidneys to the ascending and descending colon, and that traction of the colon may influence its descent. Reversed peristaltic action occurring intermittently in the ascending colon, which does not take place in the descending, is believed to have an influence, and the peritoneum over the left kidney is said to be thicker. The fact that the tail of the pancreas lies in front *of the left kidney* seems to me to have some bearing on the question. Absorption of the fatty capsule is probably another factor.

The peculiar position of the right kidney and lessening of intra-abdominal pressure seem to be the chief causes of its more frequent descent.

With gastroptosis we have also a relaxation of the gastro-intestinal musculature and of all the peritoneal ligaments. Changes in the position of the stomach and in its secretory and, at times, in its motor functions account for the gastric disturbances. The secretory function one might expect to be influenced by circulatory disturbances following displacement of the organ.

Associated are changes in the *position of the duodenum productive of stasis*, and which *readily account for gall-bladder symptoms simulating stone*, so often attributed to nephroptosis. Similar disturbances in the intestine, constipation, diarrhea, mucous colic, or chronic appendicitis can thus be accounted for. There is a relaxation of the broad ligaments and with it ovarian and uterine descent, and even descent of the pelvic floor, with dysmenorrhea and various pelvic symptoms. I do not agree with Edebohls' theory of compression of the superior mesenteric vein by the kidney as a cause of congestion of the appendix.

Occasionally, Dietl's crisis from torsion of the kidney pedicle and, rarely, nephritis or hydronephrosis occur. More rarely the kidney may become adherent to the gall-bladder or appendix. In addition, circulatory disturbances and marked neurasthenia, the latter due chiefly, I believe, to auto-intoxication, are present; and from the severe type of splanchnoptosis we have the symptoms complex of Glénard's disease—all of which the " kidney experts " attribute to nephroptosis.

**Etiology.**—We must remember that the vertical stomach is the fetal position of the organ and every infant is born with it in this position. After a few weeks or months, through the weight of the food and the action of the diaphragm, the position of the stomach becomes normal. Occasionally, it may remain vertical, but I believe this is true more especially in those suffering from the congenital constitutional defect, to which I shall refer.

The causes of gastroptosis may be divided into congenital and acquired:

1. Congenital constitutional defect, the long narrow thorax, with the diaphragm and liver pushed down. In these, splanchnoptosis is a constitutional defect. Stiller's floating tenth rib is usually present. Butler,[1] from more recent investigations, holds that the floating tenth rib is rare in adults; though fully 50 per cent. of all children have a movable tenth rib, though it is rarely unattached. Mobility of this rib is not a stigma of enteroptosis in children. Butler holds that the ptoses are usually first noted at the period of puberty. 2. Other skeletal deformities, spinal curvature, rickets, kyphosis, kyphoscoliosis. 3. Intrathoracic pressure on the diaphragm from effusions, tumor, etc. 4. Tumors of the liver. 5. Leukemic enlargements of the spleen. 6. Tumor of the pylorus or adhesions (gastric). 7. Tumor of the colon or adhesions. 8. Chronic dilatation of the stomach. 9. Compression of the thorax by tight lacing, poor corsets, tight waist-bands, etc. 10. Relaxation of the abdominal muscles and diminution of intra-abdominal pressure. This may result from rapid emaciation in acute diseases, with degeneration of the muscular tissue; or the same condition in longer chronic wasting diseases, or *from loss of weight and muscular tone from any cause.*

Landau's disease, splanchnoptosis following confinement, is fairly frequent. Emptying of the uterus produces a sudden diminution in intra-abdominal tension, and the tendency of the viscera is to fill the vacuum previously occupied by the uterus. The abdominal walls are lax and thin from uterine pressure. The accoucheur is often to blame for not properly supporting the relaxed abdomen.

Tapping of ascites, with removal of all the fluid, may produce a similar condition; or removal of large tumors.

*Sex.*—Meynert found in 50 girls, aged twelve, 50 per cent. gastroptosis, and about 80 per cent. females in his gynecologic clinic to 5 per cent. males among adults.

From the study of various statistics it can be estimated that from about 20 to 25 per cent. of women complaining of digestive disturbances are affected with movable kidney and enteroptosis. Unquestionably the percentage of gastroptosis among all women, including those who complain of no symptoms, will average at least 15 per cent in our city population. The advocates of nephropexy find nephroptosis, disregarding the other ptoses, in 20 to 33 per cent. of all women, a satisfactory surgical viewpoint.

The improvement following *promiscuous nephropexy* in gastro-intestinal and other symptoms can be often imputed to the post-operative rest in bed and to the increase in fat by proper feeding.

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[1] Enteroptosis in Children, Jour. Amer. Med. Assoc., Dec. 31, 1910.

The ratio of males complaining of digestive disturbances, with enteroptosis and nephroptosis, is about 2 to 3 per cent. The ratio in women is 8 or 10 to 1 male.

Glénard finds a lower ratio, 70 women to 30 men in 100 cases.

*Age.*—The most frequent age is from eighteen to forty, though between fifty to sixty the condition appears most marked.

**Symptoms.**—Gastroptosis may exist without the production of any symptoms; while, on the other hand, it may be present with those of a mild character, or may finally present the aggravated type of Glénard. The following symptoms, in part or whole, may be present: Some cases complain chiefly of nervous, cardiac, gastro-intestinal or pelvic disturbances, or of special organs, such as the kidney or liver.

There are usually anemia, a feeling of weakness, or faintness and fatigue on slight exertion and backache. The appetite in some cases is poor, while in others quite good.

Some patients have the symptoms-complex of hyperchlorhydria, while others complain of belching and discomfort immediately after eating. There are usually marked and obstinate constipation, rarely diarrhea; at times intestinal catarrh or mucous colic. Flatulence is present. They have headache, are frequently nervous and hysteric, and at times neurasthenic. There is often a feeling of weight or bearing down in the abdomen, which is relieved by proper support. Menstrual disorders are frequently present, dysmenorrhea quite often. At times the pain and discomfort are focused in the kidneys, especially the right; and in addition they may have attacks of Dietl's crisis. Pains in the region of the liver and gall-bladder occur in some, and there may be attacks of pain resembling gall-stones and occasional jaundice. There may be iritability of the bladder, with frequent inclination to urinate, and pains in the ovaries and appendical region. Various sensitive points are often found in other regions of the abdomen. Palpitation is frequently present and occasionally attacks of tachycardia may occur.

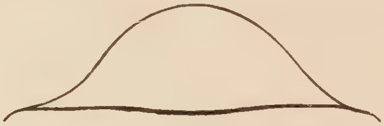

Fig. 185.—Abdominal projection; lead-tape outline between anterior superior spines; curved line, when standing; flat line, when lying on back (from Gallant).

**Physical Examination.**—*Inspection.*—These patients are usually thin and slender; the abdominal walls are generally flaccid. The form of the patient is angular and the muscles are thin. There is a concavity between the costal arches in the epigastrium from the ensiform to the umbilicus; and in some a vertical median sulcus between the recti muscles wider above than below.

In the dorsal position the abdomen may be flattened below and bulge laterally; and when the patient is erect the epigastrium becomes more depressed; while the hypogastric regions from the umbilicus to the symphysis and the pubic region markedly bulge forward and outward (pot belly). Fig. 185 shows this clearly.

*Palpation.*—Diastasis (separation of the recti muscles) can be readily appreciated. Stiller's floating tenth rib is present in some cases. Marked pulsation of the abdominal aorta is often met with as it is uncovered by the stomach. Movable kidney of varying degrees can be readily appreciated, and *this, taken in connection with the splashing sound* found below the normal position of the lower border of the stomach, is pathognomonic of gastroptosis. The corde oblique transverse is generally found to be the pancreas, which may also prolapse.

*Splashing Sound.*—This is the best method to determine the lower border of the stomach and has been thoroughly described.

If no splash can be originally detected, create it artificially by giving water, or, if required, add a little Vichy, or tartaric acid and sodium bicarbonate.

*Inflation* of the stomach with air or $CO_2$ will settle doubtful cases, as the upper border is then to be seen on inspection, and percussion is an aid.

*Gastrodiaphany* is an accurate method, but usually unnecessary.

*Percussion.*—There is at times dulness or flatness in the epigastrium when the stomach is markedly depressed, the liver descending in such cases. It is difficult to differentiate by simple percussion unless $CO_2$ distention has also been employed. The scratch method is of assistance.

**Gastric Secretion.**—*Examination of the Gastric Contents.*—Ewald's test-breakfast should be employed and gastric analysis made in every case. Hyperchlorhydria, hypochlorhydria, or, more rarely, achylia gastrica (functional) may be present. Rarely the secretion is normal, and then usually in the cases found accidentally, presenting no symptoms.

I agree with George R. Lockwood to this extent, that in many cases no evidences of fermentation can be found on test, and the gas also may be odorless. In hysteric women some of the air is swallowed. On the other hand, in some patients with associated marked dilatation (the latter probably being primary), such as I have seen among the nervous and insane at the Manhattan State Hospital, marked fermentation has been found.

The treatment is modified by the gastric findings. It is evident, in some cases, that the secretory conditions are influenced by the misplacement, since Graham-Rogers, at the Ward's Island Clinic, found that in 4 out of 7 cases (6 of hyperchlorhydria and 1 of hypochlorhydria) improvement followed the use of Rose's belt[1] alone, without medication or special diet.

**Motor Functions.**—Motor insufficiency[2] is undoubtedly present in some cases. On the other hand, many cases exist with few or any gastric symptoms, and though there is relaxation of the musculature of the stomach, compensation takes place probably by relaxation of the pyloric ring, so that the contents of the stomach enter the intestine within the normal limit of time. *Per contra* on some occasions, or from some cause, this compensation may fail and then symptoms develop. Motor functions appear only slightly diminished in other cases, or even normal, as just explained.

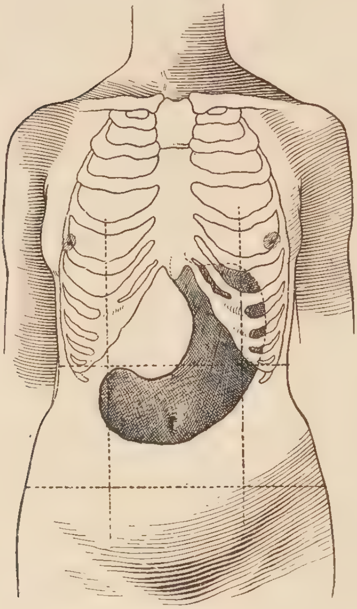

Fig. 186.—Slight gastroptosis.

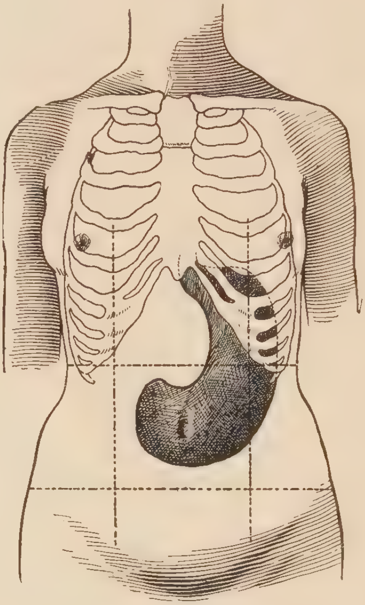

Fig. 187.—Gastroptosis.

This is the probable explanation for those cases which have existed for years without symptoms and in whom they suddenly develop.

**Stomach and Small Intestine.**—Gastroptosis may occur in various forms and degrees, semi-oblique of different degrees, looped, or, more rarely, crescentic, and even the vertical stomach (Figs. 186–190). In some cases we may have primary dilatation and then ptosis. These illustrations represent gastrodiaphany of various cases.

Mainert and Holzknecht[3] point out that gastroptosis is always accompanied by lengthening of the stomach. Bonninger holds that the lengthening can be demonstrated by the $x$-rays and is brought about by a longitudinal growth of the body.

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[1] Rose and Kemp, Atonia Gastrica, p. 124.

[2] Ibid., p. 79.

[3] Med. Press and Circular, Feb. 23, 1910.

As far as I can determine, from physical examination, post-mortem, and operative cases, especially in the marked semi-oblique or in the vertical stomach, there seems to be a *straightening of the pyloric curve* and some dilatation of the pyloric end and of the duodenum, probably with relaxation of the pylorus. The supports of the duodenum are relaxed and it crosses the spine at a lower level. I believe there is some relaxation with descent of the pancreas, and also relaxation of the mesentery and descent of the rest of the small intestine.

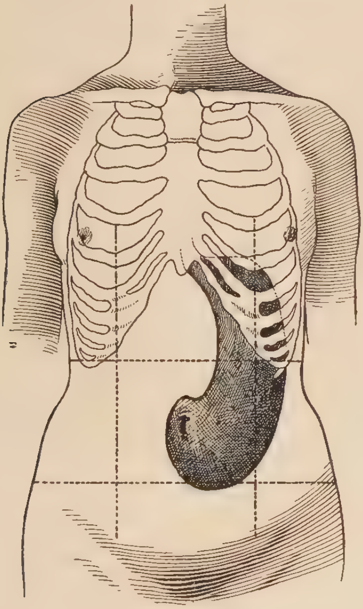

Fig. 188.—Vertical stomach.

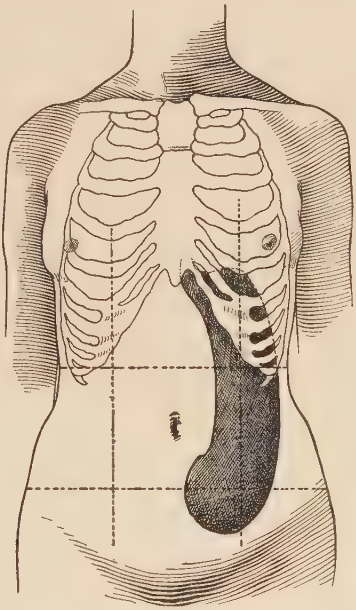

Fig. 189.—Vertical stomach (extreme).

The duodenal distention is to be expected from gravitation of the stomach-contents, and as there is relaxation of the mesentery I am skeptical of the so-called "chronic mesenteric traction" with dilatation as a result.

Gastroptosis should favor acute dilatation of the stomach, on the mesenteric traction theory, but I have never seen such a condition associated with acute ectasy.

**Large Intestine.**—*Enteroptosis.*—Descent of the transverse colon is most common (Fig. 191); it can be demonstrated by inflation with air or water, or with bismuth and $x$-rays, and they may help in differentiating between cases of redundant sigmoid, transverse colon of exaggerated length, and displaced and angulated flexures which may on rare occasions require special operation. Undoubtedly there may be relaxation, with changes in the position of the sigmoid or even of the descending colon. I have seen a case of this type recently.

**Nephroptosis.**—It seems important to further discuss this condition, though I have already described many of its features. Movable kidney from traumatism or straining, and the congenital floating kidney with a long mesonephron, constitute, I believe, a comparatively small percentage of all cases—in my own opinion, about 5 per cent.; while Meynert places them at 10 per cent. The balance are concomitants of enteroptosis (gastroptosis). It is most common on the right side.

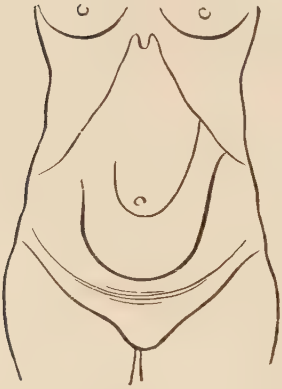

Fig. 190.—Crescentic form of gastroptosis.

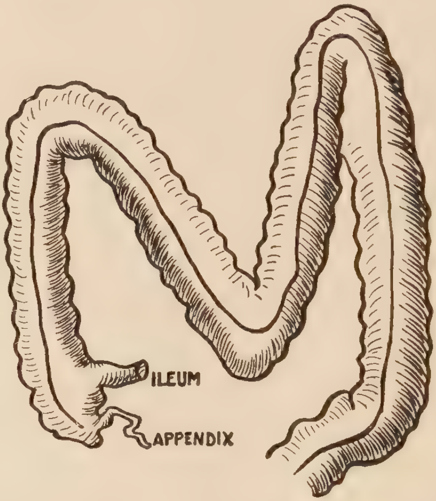

Fig. 191.—Enteroptosis.

Einhorn, as well as many other authors, recognize movable kidney as an essential symptom of enteroptosis.

Nephroptosis occurs at least six or seven times more frequently in women than in men.

Nephroptosis exists in about 15 per cent. of all women examined, associated with gastroptosis; but in many cases no symptoms are present. *No worse judgment can be shown than to tell a patient that she has a movable kidney.*

Edebohls finds it in 20 per cent. of his cases, disregarding associated ptoses, and some even place it at 33 per cent.

The normal kidney is slightly movable during respiratory movements. The most accurate method of kidney palpation I have already described.

Glénard classifies four degrees of movable kidney:

*First Degree.*—The lower pole of the kidney is palpable on deep inspiration and slips back on expiration. It cannot be arrested.

*Second Degree.*—The body of the kidney can be palpated and arrested, but not the upper border.

*Third Degree.*—The superior border of the kidney can be palpated.

*Fourth Degree.*—The entire kidney is palpable and it may be found in various regions of the abdomen, near the gall-bladder or as low as the appendical region.

I have already referred to the various symptoms attributed to movable kidney, such as dilatation of the stomach due to pressure on the duodenum; jaundice; gall-stone symptoms or stasis with the production of stone; gastro-intestinal and pelvic disturbances; chronic appendicitis as a result of congestion from compression of the superior mesenteric vein against the pancreas and spine (in 20 per cent. of cases, according to Edebohls).

All these symptoms are referable to splanchnoptosis.

Goelet believes that in 75 per cent. of nephroptosis of the third degree, or beyond, there is a pyelonephritis or interstitial nephritis.

In 20 cases of nephroptosis of the third degree or more (with enteroptosis) at the Manhattan State Hospital, I requested LeRoy Broun to examine the gynecologic conditions, and in no case could any connection be found between the prolapsed kidney and the genital organs. Ferd C. Valentine[1] and Terry M. Townsend, as a check, made special examinations of the genito-urinary tract and urine, but found no evidence of nephritis or pyelonephritis in these 20 cases.

Such examinations *should not* be made immediately after palpation, as Menge demonstrated that albuminuria appeared directly thereafter.

Schreiber[2] showed this in 39 out of 42 cases examined within ten to fifteen minutes after palpation, and that it *sometimes lasted hours.* Renal epithelium, red and white cells, but no casts were found. He believes it will furnish a *method of differential diagnosis*, as if, on palpation of a doubtful mass, this condition occurs, it will prove to be kidney. Of course, nephritis or pyelonephritis may occur in some cases; and after differential ureteral catheterization, if both kidneys are prolapsed, nephropexy is indicated in the diseased prolapsed organ.

Hydronephrosis, or adhesion of the kidney to the appendix or gall-bladder, may also occur occasionally.

In some cases more marked or severe symptoms may be *attributed to the movable kidney:*

(1) There may be a weight or special feeling of traction on that side, increased on standing or walking, and lessened in the recumbent posture.

(2) The kidney may be increased in size, tender on pressure, and there may be pain and tenderness in the lumbar region, frequent urination, and burning headache.

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[1] Kemp, Amer. Jour. of Urology, Jan., 1906.

[2] Zeitschrift für klin. Med., vol. lv, No. 3.

(3) Dietl's crisis, probably due to torsion of the pedicle, will produce severe abdominal pain, chills, nausea, vomiting, fever, and even collapse. The urine may be high colored and blood be present. Abdominal support should first be tried systematically in these conditions before operation is advocated.

**Floating Liver (Movable Liver).**—Osler claims that in a considerable number of cases *there is a mistaken diagnosis.* One anomaly is the tilting forward of the organ, so that the anteroposterior axis becomes vertical and not horizontal, and a considerable part of the surface of the right lobe is in contact with the abdominal wall.

In one type of lacing liver, the anterior part of the right lobe is greatly prolonged, a shallow transverse groove separating it from the rest of the organ.

A slight grade of mobility (floating liver) is found in enteroptosis, but the cases reported are comparatively few.

In some cases the upper surface may lie below the costal margin. G. E. Graham has collected 70 cases. The condition is rare in men.

In some cases of enteroptosis with hepatoptosis, the symptoms may be fairly marked in the liver, there being pains in the hepatic region radiating toward the back and at times of a tearing character. There may be some local tenderness and attacks similar to hepatic colic, though usually no jaundice.

Einhorn[1] described several groups of cases, dyspeptic asthma among them, but from my own point of view this condition seems to be a part and parcel of the general syndrome "enteroptosis," with marked local symptoms, such as pain or colic, in some cases. In extreme cases the liver may drop down so that the upper surface is below the costal margin.

**Gall-bladder.**—In cases of marked gastroptosis, gall-bladder symptoms simulating biliary colic at times occur. With the descent of the stomach and associated torsion, or descent of the duodenum, there is probably interference with the passage of the bile. It is interesting to note that after proper abdominal support of the prolapsed organs, the symptoms of gall-stone colic will disappear. E. Gallant reports a number of cases.

**Cardioptosis.**—In my own experience, ptosis of the heart is present only in marked cases of splanchnoptosis, especially where there is skeletal deformity. Einhorn finds it associated with floating liver. The latter is more frequently a concomitant of skeletal deformity, I believe, and hence the two conditions are associated. Movable spleen may occur from below the rib even into the pelvis. There may be dragging pains in the side, torsion of the pedicle, swelling of the organ, with pain and fever associated.

**Diagnosis.**—Curiously enough, the majority of writers pay chief attention to nephroptosis, refer to the stomach as dilated (a result of kidney pressure), and do not differentiate between gastroptosis and dilatation of the stomach. It is the position of the upper border which determines ptosis, but the following symptoms will usually settle the diagnosis. The peculiar conformation of the abdomen heretofore described, the separation of the recti, *movable kidney, and the determination that the lower border of the stomach is abnormally low,* are sufficiently diagnostic of gastroptosis.

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[1] Med. Rec., Sept. 16, 1894.

Inflation or gastrodiaphany may be necessary in doubtful cases to settle the question.

**Glénard's "Belt Test."**—If one stand behind the patient and, encircling him with the arms, lift up and support the lower protuberant abdomen, and this gives relief, it suggests enteroptosis.

**Prognosis.**—The cases of congenital type with funnel thorax or other skeletal deformities are the most unfavorable to treat; but even in these much can be done.

The acquired type presents a favorable prognosis as to absolute cure, though some tend to relapse if continuous care, as to regulation of mode of life, exercise, and diet, is not kept up. I have seen a number of cures, as has every other observer.

**Treatment.**—*Prophylaxis.*—Much can be done to prevent the acquired type of gastroptosis. Improperly made corsets compress the thorax or waist. Tight lacing and the use of tight bands around the waist are productive of ptosis. These should be avoided.

In my opinion, most of the Landau (postpartum) cases of splanchnoptosis are absolutely preventable, the fault frequently being due to the physician in attendance, the patient's abdomen not being properly supported, and she also being allowed to leave the bed too soon. There is a marked thinning and weakening of the abdominal wall during pregnancy, and after delivery immediate attention should be given to its support.

For several years Douglas H. Stewart, of New York, has been employing as a routine postpartum method of support (at my suggestion) Rose's plaster strapping, reinforcing it with lateral soft-rubber strips (an idea of his own). He states that it supports the organs perfectly, prevents abdominal relaxation, and enables the patient to sit up in bed earlier, with resulting improved drainage of the uterus, and to be about at an earlier period.

Bassler[1] treats of the necessity of abdominal support after confinement, and describes methods of strengthening the abdominal muscles and the value of forced feeding.

**Treatment of Existing Gastroptosis (Splanchnoptosis).**—There are three chief principles involved:

(1) The support and strengthening of the abdominal muscles, which also *increase intra-abdominal pressure.*

(2) The increase of intra-abdominal pressure, by reducing the

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[1] Prophylactic Measures Against the Development of Landau Cases of Visceroptosis, Therapeutic Gazette, Sept. 15, 1907.

volume of the abdominal cavity through accumulation of fat, and thus lifting up the stomach.

(3) Correction of the gastro-intestinal disturbances and the toning up of the nervous system.

(1) *Abdominal Support.*—The selection of a proper apparatus for mechanical support to the abdominal muscles, which at the same time will increase intra-abdominal tension, is the first indication.

I have employed, by preference for some time past, adhesive plaster strapping in the form of the belt devised first by Achilles Rose. I have already referred to the superiority of moleskin, first suggested in this country by me after numerous experiments. The Z. O. type should be used by the method already described. The support afforded is continuous, should never be kept on over three to five weeks, and on signs of loosening, a new belt should be applied; the patient, between belts, taking a full bath and employing talcum powder over the surface during the twelve to twenty-four hours' intermission. My longest case wore the belt fourteen months, winter and summer, it being reapplied every four to six weeks, with a gain of 44 pounds and perfect cure.

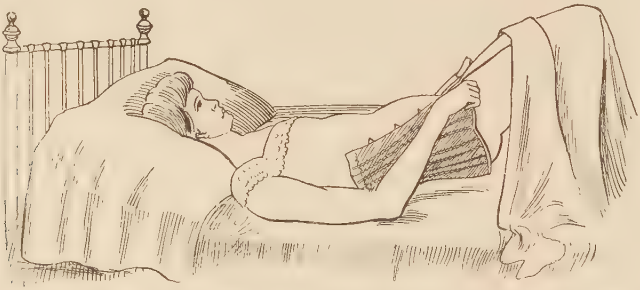

Fig. 192.—The proper way of adjusting the corset (after Gallant).

Some of the other types of adhesive strapping belts may be employed if the wide plaster necessary for Rose's belt cannot be procured. Pressure is exerted by the belt, from the symphysis to the umbilicus in front, the intestines are forced up, and hence the stomach; and the increased pressure aids in holding back the kidneys.

Next in value to adhesive plaster is the Gallant corset, which also exercises upward pressure and support below, and is loose above. The method of its application is shown in Fig. 192.

The La Grecque corset (Van Orden's), recently devised, is an excellent appliance. It well supports the spine and pelvis, is made in a single piece behind, and is subdivided in front, as in Fig. 193, *A*. In Fig. 193, *B*, is shown the corset after application. It should be applied in the dorsal position, like the Gallant corset. The pressure is exerted like Rose's belt, from the symphysis to the umbilicus, and it is loose about the thorax and upper abdomen. Of late I use the adhesive strapping for several months and then follow it with this corset. The indications are the same as for Rose's plaster.

If the patient will not consent to these methods, the silk abdominal bandages, as previously illustrated, are useful. In male cases Rose's belt and, later, the silk belt are indicated.

Supports formed with pads for special organs are unscientific.

Kilmer's belt, drawn snug below and lax above, or the Van Valzah-Hayes belt can be employed.

*Exercise.—Massage.—Gymnastics.*—There are marked inconsistencies in many of the recommendations as to active exercises, flexion, and extension of the body by elevation of the legs, gymnastics, etc., to strengthen the abdominal muscles. There is no better way to take off abdominal fat and to reduce omental fat than by these means; and in the cases with marked weight reduction it is impossible to put on fat if excessive exercises are employed.

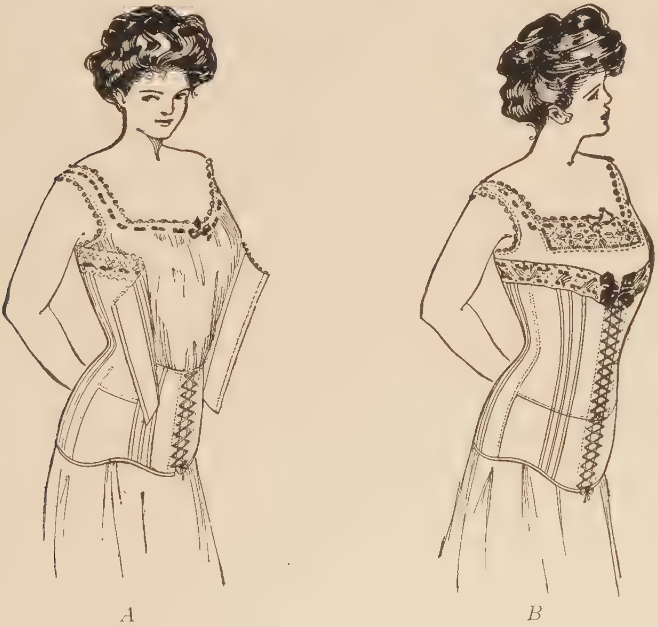

Fig. 193.—La Grecque corset: *A*, Lower segment of corset; *B*, corset after adjustment.

In moderation and properly directed, they are of service later when the weight has been increased. Driving or moderate walks in some cases are useful.

*Massage.*—On the other hand, gentle abdominal massage and often general massage of mild type, taken several times a week, will improve the muscular tone. The use of light cannon-ball massage of the abdomen, taken five minutes once or twice a day, or mild vibratory massage, at home in bed, with a good vibrator, I have found of service.

In the severe type of case, absolute rest in bed for three to six weeks with the Rose's belt applied, forced feeding, and mild massage, etc., give the best results; and if the patient is very much prostrated I omit massage at first.

2. *Increase of Abdominal Pressure through Diet by Fat Accumulation.*—*Diet.*—There are certain general principles we must follow:

If there is hyperchlorhydria, plenty of albuminous foods and fats and diminished starchy foods should be given; frequent feedings; avoidance of acids, spices, and alcohol. Often alkalis are required.

If hypochlorhydria or achylia, little meat and abundance of carbohydrates and fats, with dilute hydrochloric acid, stomachics, etc.

Not more than 8 ounces (250 c.c.) of fluid should be taken at a time and thorough mastication of the food should be enjoined. If the case is not confined to bed, a rest of fifteen to thirty minutes after each meal, if possible, is advisable. The patient should take the three chief meals daily at—

8.00 A. M.,
1.00 P. M.,
6.30 P. M.,
with intermediate feedings at
10.30 A. M.,
3.30 P. M.,
and often at
9.30 P. M.

*John Russell's method*, as employed in tuberculosis, is of value in some cases. The foods especially of use to increase weight and which can be employed for the interval feedings are:

Milk, raw eggs, 1 ounce (30 c.c.) cream in 8 ounces (250 c.c.) milk, koumiss, matzoon, bacillac, lactone-buttermilk, crackers or bread with plenty of butter, and sanatogen (preferably flavored).

Raw eggs can be given, beaten up in milk. It is well to start with 1 or 2 daily, and increase gradually to 6 or 8 per day. They possess considerable value. One could give, for example, at the intermediate feedings:

10.30 A. M.—Milk, ℥viij (250 c.c.), with 2 raw eggs.
3.30 P. M.—Koumiss, ℥viij (250 c.c.).
9.30 P. M.—Same as at 3.30 P. M., or milk with cream, and vary the methods.

Two soft-boiled eggs can be given for breakfast.

Green vegetables and raw or cooked fruits should be given for constipation, depending on the gastric conditions. Strict attention to the bowels is imperative.

I have seen the stomach elevated 3 or 4 inches by the increase of fat.

*Electricity.*—In the ambulant cases the use of static electricity will prove of service with some patients to improve the general muscular tone, or it may be employed after the rest cure has been completed. The external application of galvanic or faradic electricity I have used with good results in some cases, but never remove the belt.

Intragastric faradization has been recommended to improve the atony of the stomach, and galvanization for the pain. I have had patients bitterly complain of the intragastric method; one case notably had been treated thus systematically, with increasing nervous symptoms being produced and marked nausea, stating she was worse after each treatment. The simple method of external application seems to me advisable in those cases.

*Massage of the Kidney.*—Several methods have been recommended for treatment of the painful movable kidney.

The operator, sitting on the side of the kidney to be massaged, places the left hand (if it is the right kidney) on the lumbar region, so that the organ rests on the finger, the thumb being supported by the ribs. With the finger-tips of the right hand he pushes on the kidney from in front and gently kneads it.

Brandt places the patient in the lithotomy position, and, having replaced the kidney, places both hands in front under the margin of the ribs, and makes vibratory movements backward and upward, allowing the finger-tips to slip around to the back. The patient aids in securing this position by lifting his buttocks.

As palpation *alone will cause albuminuria, I do not approve of these methods, but merely refer to them.* It is sufficient to replace the kidney.

*Elevation of the Foot of the Bed.*—This method may be employed in those cases taking the rest cure who do not wear an abdominal support continuously. Elevation of the buttocks may be substituted. They are unnecessary when Rose's belt is employed.

Iron and arsenic are indicated in all cases, such as—

Iron tropon, ʒj (4.0), t. i. d.; or
Peptomangan (Gude), ʒj to ij (4.0–2.0); with
Fowler's solution of arsenic, ♏iij to v (0.177–0.296 c.c.), t. i. d.,
or any good combination of iron and arsenic.

An excellent pill is Blaud's iron, 5 grains (0.3), in which sodium arsenate, $\frac{1}{60}$ to $\frac{1}{30}$ grain (0.00108–0.0021), has been incorporated.

If no hyperchlorhydria is present, strychnin, $\frac{1}{60}$ to $\frac{1}{30}$ grain (0.00108–0.0021), can be included.

Hyperacidity should be treated with the alkalis.

Hypo-acidity with nux, strychnin, hydrochloric acid, and stomachics.

Bromid, 10 grains (0.6), or veronal, 5 to $7\frac{1}{2}$ grains (0.33–0.5), sulfonal, 10 grains (0.6), or trional, 10 grains (0.6), are valuable for sleeplessness, given an hour before retiring. Midonal, 5 grains (0.3), is useful.

*Hydrotherapy.*—The Scotch douche, one to two minutes over the abdomen, in convalescent cases has rendered service.

The Priessnitz compress aids to relieve pain.

A glass of cold water, Vichy or Carlsbad, on rising helps the bowel action.

*Dietl's Crisis.*—Apply heat to the kidney; employ fluid diet; elevate the foot of the bed; replace the kidney.

Codein, $\frac{1}{4}$ to $\frac{1}{3}$ grain (0.016–0.022), or morphin, $\frac{1}{8}$ to $\frac{1}{4}$ grain (0.008–0.016), may be required, by hypodermic.

*Constipation.*—Olive oil enemata; cascara; compound rhubarb, purgen, and regulin,[1] if required, are excellent.

*Complications*, such as mucous colic or catarrhal colitis, must be appropriately treated.

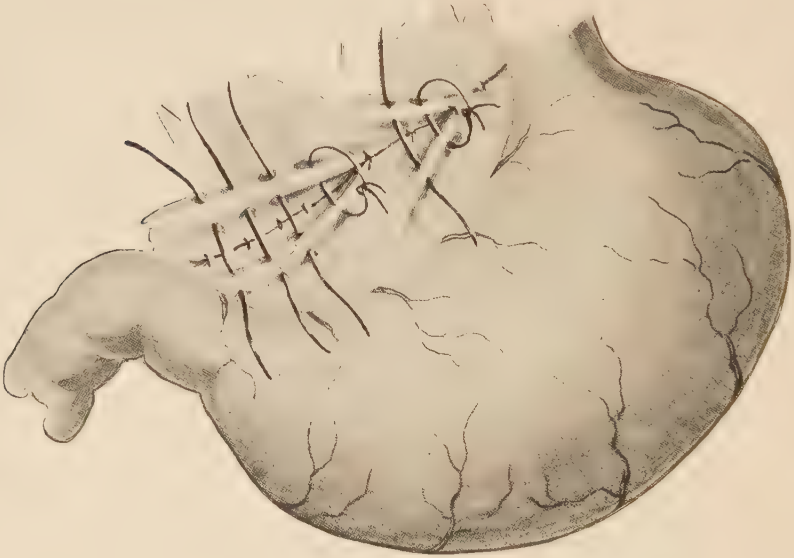

Fig. 194.—Beyea's operation; suture of the gastrohepatic omentum to secure elevation of the stomach in gastroptosis. The first layer of sutures completed, the second and third being introduced (Moynihan).

*Surgery.*—In splanchnoptosis, if all medical treatment after a year conscientiously applied prove a failure, *suture* of the recti (abdominis) and thus tightening the abdomen, as advocated by Robert T. Morris and by Charles Codman,[2] is the most scientific procedure.

If there are nephritis, pyelonephritis, or intermittent hydronephrosis confined to the prolapsed kidney, then nephropexy is indicated. After repeated attacks of Dietl's crisis, when medical measures afford no relief, nephropexy may be advised.

Hydronephrosis or pyelonephritis may be of such a character as to require nephrectomy.

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[1] See under Constipation for other remedies.

[2] Med. Rec., Oct. 19, 1907.

Operations on individual organs *I am opposed to* unless there be marked gastric dilatation *with the ptosis.*

Occasionally a redundant sigmoid may require sigmoidopexy, or it may be necessary to hitch up an angulated colon. Shortening of the lesser omentum (plication), and thus elevating the stomach, as devised by Beyea[1] is depicted in Fig. 194. When these sutures are drawn and tied, the stomach is elevated into its normal position.

Gastroplication, shortening of the lesser omentum, attaching the lesser curve of the stomach to the abdominal wall, suture of the transverse colon to the abdominal wall, sewing the greater omentum to the abdominal wall, have all been recommended.

*Gastro-enterostomy* may be indicated in gastroptosis with ectasia and marked fermentation which does not respond to medical treatment. The spleen has been stitched up or removed if inflamed.

*Duodeno-enterostomy* has been advocated by Byron Robinson, but I see no indication for it.

Elliot[2] recommends anchoring the liver in hepatoptosis.

In *rare cases*, gastroplication, with shortening of the lesser omentum; or the latter, with gastro-enterostomy, might be necessary.

[1] Jour. Amer. Med. Assoc., March 5, 1910.

[2] Med. News, Nov. 12, 1904.

## CHAPTER XVII

# NERVOUS AFFECTIONS OF THE STOMACH

Gastric neuroses may be defined as functional disturbances of the stomach without any discoverable anatomic basis, there being no organic lesion of the organ.

**Etiology.**—These cases have either inherited a nervous constitution or, through indiscretions, have brought about a condition of nervous prostration. Sometimes the gastric disturbances have apparently a reflex origin, depending on disturbances in remote parts of the body, as in the sexual organs, *appendix*, eyes, etc., and these factors must always be searched for.

For example, as a reflex from appendicitis, we may have hyperchlorhydria with or without hypersecretion, pylorospasmus, hypochlorhydria, excessive vomiting, achlorhydria hæmorrhagica gastrica (symptoms of chronic gastritis accompanied by hemorrhage apparently from erosions), and apparently symptoms of gastric ulcer. Graham[1] and Guthrie cured 89 out of 115 patients with gastric symptoms by appendectomy, and secured improvement in 20 others.

They are divided into sensory, motor, and secretory neuroses, and may appear separately or in combination. They occur most frequently in women from puberty to menopause, and quite frequently at these special periods. In men they appear most often during middle life and most frequently in the higher classes. There is probably a predisposition to the condition. Worry, extreme mental exertion, excesses, and excitement may be predisposing factors. Organic lesions of the stomach must be excluded.

**Classification.**—Neurosis of the stomach may present the appearance of some primary disease, or may be one of the symptoms of hysteria or neurasthenia, or may *be a reflex symptom due to disease of some other organ.* In cases of this last description, though there is no organic change in the stomach and the disturbance in this organ is purely reflex, there is an anatomic cause elsewhere. They should, strictly speaking, be *differentiated* from the pure gastric neuroses, since treatment of a distant diseased organ may cure an *apparently pure gastric neurosis.* This emphasizes the necessity of thorough examination. *Pure gastric neurosis I believe rare.*

**Peculiarities.**—Generally the nervous system shows more or less deviation from normal conditions. Leube and Boas have probably best described the condition. There are headache, mental depression, lack of energy; at times fear, palpitation, dyspnea, and sweating. On the other hand, there may be excitement and sensitiveness. There are an increase or diminution of reflexes, local hyperesthesia, paresthesia, or anesthesia. Polyuria may or may not be present. Some patients remain in comparatively good condition, while others emaciate.

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[1] Jour. Amer. Med. Assoc., March 19, 1910, p. 960.

The digestive disturbances are usually *independent of the quantity and character of the food*, and are not always connected with the act of digestion. Errors in diet are often not followed by an exacerbation of the symptoms, and sometimes they occur when the simplest food has been taken.

The gastric secretion and motor functions are often variable. We may find hyperchlorhydria at one time and normal secretion at another, or, conversely, normal conditions may alternate with hypo-acidity or anacidity. At times the motor functions may be normal, at others, diminished.

The condition of the bowels also varies; there may be constipation alternating with diarrhea; or the bowels may be regular and a sudden diarrhea appear.

Climate and surroundings may influence the condition.

## SENSORY NEUROSES OF THE STOMACH

There may be abnormal sensations more or less general in character *external to the stomach*, such as regarding hunger, appetite, or abnormal sensations within the stomach itself.

Among the abnormal sensations of appetite are bulimia (or canine hunger); parorexia (perversion of appetite); polyphagia (excessive eating); akoria (absence of satiation); anorexia (loss of appetite).

### BULIMIA

Bulimia, hyperorexia, or canine hunger (cynorexia) denotes a marked increase of the sensation of hunger which may occur in attacks, either as an independent neurosis or as a secondary affection, the result of some other disease. The attacks are paroxysmal. The hunger center is believed to be located in the medulla. This center is probably irritated when a certain degree of impoverishment of the blood takes place. On the other hand, if the latter is marked, the sensation of hunger may not be produced or may even be suppressed. The stomach is also concerned in its production, and the amount of food contained therein has an influence. For example, with hyperacidity and increased motor function, hunger is present. In a case of mine suffering from stenosis of the pylorus and dilatation of the stomach, while before operation anorexia was marked, the patient is now continuously hungry. Reflexly, the hunger center is often disturbed; this loss of appetite may occur after fright or on the appearance of food that is greasy and badly served, whereas with food daintily prepared and of pleasant odor, the appetite is increased.

The olfactories have also undoubtedly an influence on the digestion.[1] For example, it requires about 2000 cubic feet of air to pass daily through the lungs to furnish sufficient oxygen to maintain digestion. As the greater part of the air passes over the olfactory region, the contact of odorous substances may exert a marked influence. Moreover, appetizing odors may stimulate the salivary glands and the flow of the gastric juice; while repulsive smells may lessen the appetite, inhibit the secretion of the gastric juice, and lessen the motility of the stomach and the salivary gland secretion. The time at which hunger appears depends on the time the person is accustomed to eat.

Pathologically the sensation of hunger may also be stimulated or inhibited in various ways.

**Etiology.**—Bulimia may be a primary affection or may be secondary to ulcer of the stomach, hyperchlorhydria, hypersecretion, epilepsy, hysteria, neurasthenia, tumor of the brain, tapeworm, intestinal diseases, rarely to diseases of the pancreas or to diabetes mellitus.

**Occurrence.**—It is more frequent in women than in men, and most frequent from eighteen years of age to menopause.

**Symptoms.**—The patient may be in perfect health, when suddenly a feeling of intense hunger comes on which is extremely persistent and calls for food. There is frequently a gnawing in the stomach and a feeling of fear and anxiety. Unless the hunger is relieved, headache, trembling, and even fainting spells may occur. The attack may take place immediately after a large meal or come on in the night. In some cases small amounts of food will relieve the conditions; in others, enormous quantities are necessary. One patient recorded ate 23 eggs in forty-five minutes and drank 1½ quarts of milk and 1 quart of wine.

**Character of Attacks.**—They may be severe or very slight. In some cases they occur every few hours; in others they last a few days, or they may exist chronically and last for years. The periodic form is generally more intense. Hypermotility is found in some cases.

**Prognosis.**—In the secondary form it depends on the primary disease, though sometimes bulimia persists. The more frequent and violent the attacks, the worse the prognosis. Ordinarily it is difficult to give an absolute prognosis. Gastritis, atony, or dilatation may result in some cases.

**Treatment.**—If bulimia is secondary, the primary disease should be treated, such as hyperchlorhydria, tapeworm, diabetes, or neurasthenic or hysteric symptoms. For the bulimia we should give frequent light meals every two hours. The bromid of sodium, potassium, ammonium, or strontium may be given in 20-grain to ½-dram (1.3–2.0) doses two or three times daily; for example:

℞. Sod. bromid.............................. ʒiiss (10.0);
Aq. menth. piperit....................q. s. ℥iv (125 c.c.).—M.
Sig.—Two teaspoonfuls in water t. i. d. after meals.

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[1] Niles, Jour. Amer. Med. Assoc., Oct. 16, 1909, vol. liii, pp. 1274–1278.

Codein, opium, and cocain I strongly deprecate, though recommended by some. There is great danger of acquiring the habit.

Tincture belladonna, 10 minims (0.59 c.c.) t. i. d., is a good substitute.

Arsenic (Fowler's solution), 5 minims (0.296 c.c.), or sodium arsenate, $\frac{1}{50}$ to $\frac{1}{25}$ grain (0.0013–0.0026), or arsenous acid, $\frac{1}{100}$ grain (0.00065) t. i. d., are useful.

Iron can be given, also strychnin, $\frac{1}{60}$ grain (0.00108), for the nerves.

Change of climate is of value. Food should be carried by those suffering from abnormal hunger for immediate use.

## Parorexia (Perversion of Appetite)

Sometimes the appetite is manifested for special or peculiar kinds of food. There are three types:

(1) *Malacia*, a desire for spiced foods, such as for mustard, vinegar, green fruits, etc.

(2) *Pica*, a desire for substances that are not foods, such as earth, chalk, ashes, sand, insects, etc.

(3) *Allotriophagia*, a craving for disgusting and harmful substances, such as fecal matter, pins, etc.

Malacia is met with in disturbances of the stomach and neurasthenia, while the other types occur chiefly in idiots, lunatics, or severe hysteria. The treatment is of the disease which is the cause of the perversion.

## Polyphagia

Polyphagia is the demand for large quantities of food *before satiation* occurs. The cases do not feel hungry until the food is *digested*. It may occur as a primary condition in neurotics, or secondary to diseases of the gall-bladder, spleen, diabetes, or brain tumor. The attacks may be of short duration or as a chronic trouble. One case could eat 100 pounds of meat in twenty-four hours.

Disease, if present, should be treated; neurotic conditions should be corrected. The general treatment is the same as of bulimia.

## Akoria

Akoria is absence of satiety. Patients with akoria never feel *satiated*, and never know when to stop eating. It is at times combined with polyphagia. It is met with among the neurasthenic and hysteric. Bromids are of service and the general treatment of the nervous condition.

## Nervous Anorexia (Anorexia Nervosa)

Anorexia is diminution or loss of appetite, with absence of the hunger sense, so that even *aversion to food* may be present.

It occurs in most of the organic as well as in the functional disorders of the stomach, but the nervous type appears as a primary affection.

It has been attributed to a depressed condition of the hunger center or to hyperesthesia of the mucous membrane of the stomach.

**Etiology.**—Psychic shock, some mental depression, worry, a disagreeable odor, or some nauseating sight may cause *transitory anorexia* in a healthy person.

It may, however, be more persistent in hysteria, neurasthenia, and the psychoses; *morphin* and excessive smoking may produce it. It is more frequent in *women* and may result from anemia or chlorosis.

**Symptoms.**—There is at first loss of appetite, and the patient begins to eat less. These cases may first have an aversion to meat, and later to bread and butter, vegetables and all solid food, and may finally live only on fluids, sometimes on very small quantities. They often vomit at the sight or smell of food, and may, in severe cases, emaciate markedly. They will refuse to increase their nourishment ("siturgy"). The pulse becomes slow and the temperature subnormal; they become pale, cyanotic, and the eyes sunken. Such cases may even terminate fatally. This condition may be mistaken for organic disease or for phthisis, and a careful physical examination and gastric analysis should be made.

**Treatment.**—The patient should be impressed with the idea that he must take his food in sufficient quantity and eat everything put before him. He should not be questioned as to his desires.

Frequent small meals with koumiss, matzoon, bacillac, lactone-buttermilk, butter, milk, and cream should be given to improve nutrition. Raw eggs are of service; also sanatogen up to 1 ounce (32.0) daily in divided doses.

The sour milk products are of value, as auto-intoxication is an important feature in these neurasthenics.

Stomachics, such as tincture of nux vomica, 10 minims (0.59 c.c.) t. i. d., and fluidextract of condurango, 20 minims (1.184 c.c.), are of service. Fluidextract of Peruvian bark, 1 dram (4.0) t. i. d., is excellent.

℞. Tr. nucis vomicæ,
Acid. hydrochlor. dil. } ....................āā ʒij (8.0);
Comp. tinct. cinchona.................... ℥ss (16.0);
Aq. destil..........................q. s. ℥iv (125.0).—M.
Sig.—ʒj to ij (4.0-8.0) t. i. d. in water before meals.

Basic orexin, $\frac{1}{3}$ to $\frac{1}{2}$ grain (0.02–0.03) t. i. d., may prove of service. Morphin and tobacco, if they are factors, should be cut off.

Sanatorium treatment and *rest cure* are important in severe cases, especially by the Weir-Mitchell method. Isolation from the family, strict supervision by the physician, massage, and electricity are valuable.

Forced feeding (gavage) or nutritive enemata may be required. After a while the patient will be convinced she can digest food. Small quantities are given at first, and then they are increased. In milder cases change of climate is useful.

Strychnin, $\frac{1}{60}$ to $\frac{1}{30}$ grain (0.001–0.002), Blaud's iron pills, peptomangan (Gude), or iron tropon are of service. Arsenic may be added, Fowler's solution, 5 minims (0.29), sodium arsenate, $\frac{1}{60}$ to $\frac{1}{30}$ grain (0.001–0.002), glycerophosphates of soda, or Chapoteaut's glycerophosphate of lime. These remedies should be given t. i. d.

### Sensations Within the Stomach

Under *normal conditions* we do not recognize that we have a stomach, and there are no sensations after the ingestion of food. The stomach is not devoid of sensation even normally, as excessive quantities of hot or cold material are noted by internal sensations, such as after the ingestion of a large quantity of ice-cream on an empty stomach.

Gastric sensation, however, may be increased to a pathologic degree and be a source of great discomfort. We must remember that certain persons are peculiarly constituted and have idiosyncrasies to particular foods.

**Idiosyncrasies.**—For example, shell-fish, such as lobsters, crabs, and oysters, strawberries, bananas, onions, etc., will produce skin eruptions such as urticaria or erythema, and gastric symptoms such as pain, belching, pressure, and even nausea or vomiting. Talma and Einhorn have described cases having a peculiar idiosyncrasy to hydrochloric acid. I have seen several cases where the patient suffered from the symptoms of hyperchlorhydria, and yet the free hydrochloric acid found was well within normal limits. The administration of alkalis relieved the symptoms. The gastric nerves were evidently peculiarly sensitive to acid in these cases. If patients have an idioscyncrasy to special food, it must be avoided.

### Abnormal Sensations (Sensory Neuroses of the Stomach)

The nervous and hysteric at times complain of sensations of heat, cold, or of some foreign body, such as the feeling of worms, etc., within the stomach. These symptoms are not dependent on the food or upon the gastric secretion. There may be in some a feeling of constriction, cramp, or " pulsation " within the stomach. The latter is probably due in many cases to hyperesthesia and the transmission of the aortic pulsation.

*Nausea.*—There may be a nervous type of nausea in such patients, either during the fasting condition or after meals, the treatment for which should be directed to the neurasthenia. Nausea may also be reflex, as from an affection of the genital organs or in organic disease of the stomach. Einhorn recommends intragastric galvanization.

The indication, however, is to treat the hysteria or neurasthenia.

### Hyperesthesia of the Stomach

This consists in an increased sensibility of the nerves of the stomach, so that the mucous membrane is abnormally sensitive even after ordinary food is taken. It differs only in degree from gastralgia. It may be secondary to organic affections, associated with secretory disturbances, or it occurs as a primary neurosis.

**Etiology.**—It is present especially in the higher classes. Mental overexertion, excitement, alcohol, and venereal excesses, which weaken the constitution, are factors. It is associated with hysteric or nervous symptoms. Though anemia is given as a cause, hyperchlorhydria with hyperesthesia occur with this condition, and this type is, therefore, not a pure sensory neurosis.

**Symptoms.**—There is a sensation of fulness, weight, or pressure after meals, which may become real pain, and, finally, vomiting may occur. Sometimes considerable emaciation is present, as the patient fears to eat (sitophobia). Hyperesthesia occurs after excess of food or, in some, in cases of fasting. Persistent hiccough may occur with this condition.

**Physical Signs.**—The epigastrium and region of the stomach are sensitive to pressure throughout, but there is no specially sensitive area as in ulcer.

Certain articles of food, such as sugar, fat, starch, or coffee, may produce the condition. The secretory and motor functions are normal in the purely nervous cases.

**Diagnosis.**—This is based on the absence of organic disease of the stomach, the absence of hyperchlorhydria or of other secretory disturbances, and the absence of motor disturbances. With hyperchlorhydria and gastritis the gastric analysis and other symptoms will give us the requisite differential points. With ulcer, hyperacidity is the rule, and the degree of pain is dependent considerably on the character of the food, which is not *generally so in hyperesthesia;* the other symptoms also differ.

With *erosions* small bits of mucous membrane are washed out and there are secretory changes.

**Prognosis** depends on the result of the treatment of the nervous condition.

**Treatment.**—In many of these cases, as when anemia is present, secretory disturbances are associated with the sensory neurosis, as hyperchlorhydria, so that the pure sensory condition is often complicated. Iron and arsenic are necessary in such cases and, in fact, have an excellent effect. Peptomangan, iron tropon, or any good iron preparation combined with Fowler's solution, 5 minims (0.296 c.c.) t. i. d., is excellent.

The patient should be kept in bed in severe cases and warmth applied to the stomach, dry heat, or Priessnitz compresses. Fluid diet in rather small quantity should first be given. Milk and lime-water, sanatogen, broths, chicken soup, white of egg, later entire raw eggs beaten in water, calves'-foot jelly, scraped meat, zwieback softened in milk, butter, and, gradually, solid food.

Tincture of belladonna, 5 to 10 minims (0.296–0.592) t. i. d., is excellent for the pain. The use of opium and its derivatives and cocain are to be *deprecated*. Rarely an occasional dose of codein may be required.

In some cases silver nitrate, $\frac{1}{8}$ to $\frac{1}{4}$ grain (0.008–0.016), in aqueous solution t. i. d. before meals is of service.

External galvanization to the stomach is of value. Bromids in some cases are serviceable.

Baths and change of scene may be enjoined. Suggestion should be used by the physician.

## GASTRALGIA

(*Synonyms.*—Gastrodynia, Spasm of the Stomach (Gastrospasmus); Cardialgia, Neuralgia of the Stomach.)

Gastralgia may be defined as the occurrence of violent attacks of pain in the stomach, paroxysmal in character and alternating with free intervals.

**Etiology.**—We may have a number of forms of gastralgia originating from various conditions, and these must be referred to in order to differentiate them from the purely nervous type.

*Gastralgia Originating in the Stomach.*—(*a*) Connected with organic affections, such as ulcer, cancer, stenosis of the pylorus, or gastritis, with hyperacidity, hypersecretion, peritonitic adhesions, or perigastritis.

(*b*) Special varieties of food, as rich spices, or of drink, as strong coffee, or ice cream, etc., may produce it in people not accustomed to these substances.

*Spinal disease, as tabes, may cause gastric crises, or, more rarely, cerebral disease* or various types of myelitis. Absence of patellar reflex, Argyll-Robertson pupil, and Rhomberg symptom are diagnostic of tabes.

*Gastralgia as a Neurosis.*—This occurs with neurasthenia or hysteria. It may appear before the nervous symptoms are in evidence.

*Reflex Causes.*—Gastralgia of this type occurs most frequently in women. It takes place reflexly from disturbances in the female or in the male genital organs. Gastralgia may occur at the time of menstruation or in place of it.

Disease of the liver, spleen, bladder, pancreas, and ptosis of abdominal organs may be reflex causes.

*Other Causes.*—*Malarial infection* may be a cause of gastralgia. The latter may be associated with the usual symptoms of malaria or it may be substituted for the malarial cycle, appearing every day, alternate day, or third day at the same hour, as do other neuralgias.

Gouty infection, mercurial or lead-poisoning, or excessive smoking may produce it. Exophthalmic goiter, anemia, or chlorosis with malnutrition may be a cause, although the attacks in many of these cases are the result of the associated hyperchlorhydria.

*Sex.*—Most frequently in women and girls from fifteen to forty, and decreases in frequency with age.

**Symptoms.**—The attacks usually appear suddenly and occur in paroxysms, though they may be preceded by nausea, with belching and distention, headache, or dizziness. There is a sudden extreme pain in the epigastric region of a boring, tearing, burning, and constricting character. It may radiate over the abdomen or to the back and shoulders. The attacks may occur independently of eating and whether the stomach is empty or full, or at any hour (*in the nervous cases*).

The face is pale and distorted with the pain, by which the patient is frequently doubled up, as with colic, and there is inability to lie straight, and often clammy sweating. Strong pressure on the abdomen frequently relieves the pain, though it may be sensitive to lighter pressure. The gastric region is usually sunken.

There are *often hiccough* and belching, with nausea and collapse. The pulse is generally rapid and feeble, though in some cases slow.

**Duration.**—Gastralgic attacks may last from a few minutes to an hour or longer. There may be several attacks in a day, every few days, or at intervals of weeks or months.

The pain disappears suddenly and may be followed by hunger or even bulimia. In mild cases the patient may not be greatly affected and may be able to work immediately, while in the severe cases there is often prostration for several days.

**Prognosis** is favorable as far as life, and depends on the removal of the primary cause.

**Differential Diagnosis.**—The sudden onset, violent spasmodic pain in the stomach, general in character and lessened by pressure, nausea, vomiting, and headache—that these symptoms *are independent of eating and frequently occur after mental overexertion or emotional shock* and are associated with nervous hysteric symptoms—all point to the nervous type.

We must exclude the secondary form *by gastric analysis*, and by the determination *that no organic disease exists.* A careful physical examination is necessary, thereby eliminating other conditions that may cause pain in the gastric region.

*Ulcer of the Stomach.*—The pain at the height of digestion depends on the quantity and quality of food; *disappears when the stomach is empty;* intervals free from pain; hyperchlorhydria often present; circumscribed spot in the epigastrium painful on pressure and increased by it; hematemesis present in some cases, or occult blood and pus in gastric contents or stool. If these characteristic symptoms are absent it may be necessary to try one of the ulcer cures; which, if it fails, would rather point toward nervous gastralgia.

*Cancer of the Stomach.*—Pains not as severe, but more continuous, never free from pain; absence of hydrochloric acid most frequent; lactic acid and Boas-Oppler bacilli present; cachexia; age of patient; tumor often present.

*Chronic Gastritis.*—Intense pains are absent; are continuous and more a sense of discomfort after the ingestion of food; no paroxysms; mucus in gastric contents; hypochlorhydria.

*Stenosis of the Pylorus.*—Attacks of pain (gastralgia), associated with *peristaltic* unrest and vomiting. Dilatation of the stomach is present; usually hyperacid contents if benign stenosis; and lactic acid present with absence of free HCl and Boas-Oppler bacilli if malignant.

*Functional Disorders.*—*Hyperchlorhydria* and *hypersecretion;* pains disappear after albuminous food or alkalis. Gastric analysis, hyperacidity; and if hypersecretion, excessive quantity of secretion in the empty stomach of high acidity.

*Achylia Gastrica.*—Pains disappear when stomach is empty. Gastric analysis shows low acidity, 2+ or 4+; free HCl absent; rennet = 0; pepsin = 0.

*Rheumatism and Myalgia.*—Myalgia is muscular pain which may be due to exertion. In both conditions the pain is not paroxysmal; corresponds to the course of the muscles, and is relieved by abdominal relaxation; we have rheumatic history or that of over-exertion.

*Intercostal Neuralgia.*—Pain is superficial and can be traced along the intercostal nerves, which are sensitive at numerous points on pressure.

*Renal Calculi.*—Pain in the kidney (dorsal region) radiates to the ureter and bladder; testicle drawn up in the male; urine is acid and shows albumin, casts, and blood. Sand often present. Kidney is frequently tender on pressure.

*Gall-stones.*—Pain over liver and gall-bladder; deep pressure increases the pain; rise of temperature; pain passes to right and often up into the right shoulder. Head's gall-bladder zone often present. Leukocytosis frequently present.

Jaundice occurs at times and gall-stones in the stool are conclusive.

*If the motor or secretory functions of the stomach are normal,* the attack of pain—especially if it is in the right hypochondrium and *the patient has no nervous symptoms*—is probably due to gall-stones.

With gastralgia there are no pain and no fever. It is sometimes difficult to differentiate between the other conditions.

*Perigastritis.*—A high position of the stomach, with the left lobe of the liver drawn down and covering that organ, is suggestive of perigastritis when pains come on regularly several hours after meals (Kaufmann).[1]

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[1] Amer. Med., vol. vi, No. 20, pp. 792, 794, Nov. 14, 1903.

*Intestinal Colic* (*Enteralgia*).—Pain changes its position in the abdominal cavity. Passage of flatus relieves pain. Bowels are irregular.

**Treatment.**—In secondary gastralgia it is necessary to treat the primary cause.

If it is due to tobacco, it should be cut off. Malaria should be treated with quinin or Warburg; and anemia, with iron and arsenic and the rest cure. Correct a gouty tendency, if present; or treat tabes, if this be found.

Sexual disorders, ulcer, gall-bladder disease, or cancer should receive appropriate treatment.

If hysteria or neurasthenia are present, tonics, isolation, the rest cure, massage, and hydrotherapy are of value.

The galvanic current, the anode over the stomach, or intraventricular galvanization, with the cathode over the spinal column, is of service when repeated attacks of primary gastralgia occur.

*For the active condition*, if moderately severe, hot applications (dry or moist heat) and hot drinks are useful.

Tincture of belladonna, 10 to 15 minims (0.592–0.088), to relieve the spasm. Tincture of valerian, 20 minims to ½ dram (1.184–2.0), in water, or Hoffmann's anodyne, ½ dram (2.0), may be given in water with sugar. Chloroform spirits, 2 to 3 drops in water at a dose, can be administered. The bromids are also valuable.

If there is retention of food during the attack, then lavage with hot water will relieve some cases.

If the pain is unbearable, codein, ½ grain (0.032), by hypodermic, or morphin, ¼ grain (0.016), with atropin, 1/100 grain (0.00065), can be administered.

Suppositories of morphin, ¼ grain (0.016), and extract of belladonna, ¼ grain (0.016), or opium, 1 grain (0.065), and extract of belladonna, ¼ grain (0.016), every two to three hours for several doses, are useful.

### GASTRALGOKENOSIS (BOAS[1])

This is characterized by pain in the stomach when it becomes empty, and is relieved by food. It may be periodic or permanent. Frequent meals and nerve sedatives are required.

## MOTOR NEUROSES OF THE STOMACH

Under normal conditions the cardia remains closed after the process of digestion has begun, while the pylorus opens at intervals to allow a certain amount of escape of chyme. The muscular movements of the stomach mix the ingesta with the gastric juice and aid in its disintegration by churning movements. Later they propel it into the intestine. These motor functions may be irritated (exaggerated) or depressed (diminished).

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[1] Krank. des Magen, II, Feb. 418, Auflage, S. 260, Leipzig, 1901.

## HYPERMOTILITY OF THE STOMACH

(*Synonyms.*—Supermotility; Hyperkinesis; Hyperanakinesis Ventriculi—Einhorn.)

In this condition the stomach propels the ingesta into the intestines more rapidly than normally. It may be secondary to achylia gastrica or hyperchlorhydria or diseases complicated thereby, or the condition may exist as a primary neurosis.

In some cases achylia and hyperchlorhydria exist as a secretory neurosis, and with this there is the motor neurosis, hypermotility.

Supermotility may exist as a motor neurosis alone (primary neurosis), due to some nervous influence or associated with nervous conditions, and is analogous to increased intestinal peristalsis from nervous influence. In pyloric obstruction there is often actual increased motor power in the early stages, but the resistance to the exit is so increased that the final result is relative motor insufficiency. In the secondary cases hypermotility will be relieved by treating the cause.

**Diagnosis.**—Aspiration of the stomach-contents will demonstrate the condition. For example, one may find the stomach empty thirty-five to forty-five minutes after the test-breakfast, or only a minute quantity can be removed.

Einhorn suggests the employment of the gastrograph, noting an increase of the "makes" and "breaks," but this method is complicated.

Careful analysis of the gastric contents should be made to test the secretory functions, and an early aspiration would be necessary.

Hypermotility in itself does not produce any special gastric symptoms.

## PERISTALTIC RESTLESSNESS OF THE STOMACH (KUSSMAUL)

(*Synonyms.*—Peristaltic Unrest; Tormina Ventriculi Nervosa.)

This is really an exaggerated hypermotility in which the peristaltic action is unusually great, and is appreciated by the patient as a disagreeable sensation. It may even become visible through the walls of the abdomen, and waves of contraction can be seen running from left to right along the stomach, and may take from three-quarters of a minute to a minute or more. This condition usually occurs when there is obstruction at the pylorus or duodenum, with dilatation of the stomach, and is produced by the effort of the organ to overcome the obstruction.

Kussmaul first described 2 cases of peristaltic unrest which were pure neuroses of motility. The movements began early in the empty stomach and became more violent after eating. Gastroptosis was present.

Emotional shock and abuse from sexual excesses were the causes.

Mechanical forms must be excluded. In these it occurs only when the stomach is full. In the nervous cases movements take place both when the stomach is full and empty. If the patient is well nourished and the stomach is in normal position, it may be difficult or impossible to see the movements, but the patient will complain of the contractions and wave-like movements, and borborygmi and gurgling may be heard. Nausea, vomiting, and cramp-like pains may occur, especially in the obstructive type.

Physical examination and gastric analysis will determine whether the peristaltic restlessness is primary (a neurosis) or secondary.

Course and prognosis depend upon the cause and the nervous condition in primary cases.

Cases may be continuous, when they may cause loss of sleep, or may be intermittent.

**Treatment.**—This must be directed against the primary cause, such as stenosis of the pylorus, etc.

If it is a pure gastric neurosis, resulting from mental overexertion, grief, sexual excess, or nervous or hysteric conditions, we must remove these factors. Iron, arsenic, and the glycerophosphates are indicated.

Hydrotherapy, diet, and change of scene are valuable. The diet should be regulated, all irritants to the stomach should be avoided. The food should be digestible, non-irritating, and in moderate quantities at a time, especially at night.

*Locally*, heat or cold to the stomach; galvanic electricity, percutaneous or intragastric. This last is of no value in stenotic cases, but does harm.

Lavage is indicated if there are stenosis and dilatation as a temporary measure with operation for permanent cure.

## ANTIPERISTALTIC RESTLESSNESS OF THE STOMACH

In rare cases the peristaltic action is reversed, the waves running from right to left. These cases are generally of neurotic origin.

Intestinal waves are of small caliber and move in different directions in various regions, while gastric waves are of large size and move in the gastric region. These waves may extend to the intestines and colored enemata have been voided from the mouth (Osler).

Treatment should be directed to the neurosis.

## INCONTINENCE OF THE PYLORUS

(*Synonym.*—Insufficiency of the Pylorus.)

This condition was first described by de Séré and Ebstein.

It may be caused by some growth in the pylorus, keeping the opening patent, or by stenosis or other organic changes, or when the pylorus is relaxed (atonic), due to some nervous derangement, hysteria, or myelitis.

Relaxed pylorus, I believe, to be frequently associated with atonic ectasy, and that this feature accounts for the absence of pain and vomiting. It is also present in some cases of gastroptosis. The relaxation in these cases is probably compensatory. *I do not believe it to be a true incontinence*, but merely atony with weakening of the pyloric musculature, *with no regurgitation.*

Ebstein has shown that if we attempt to inflate the stomach with air or $CO_2$, it will rapidly pass into the intestine and distend it, and gastric tympany is absent. *A diagnostic feature of true incontinence is the regurgitation of the intestinal contents into the stomach.* On aspiration or lavage in the fasting condition, intestinal juice and often a considerable quantity of bile will be found. It is sometimes present on aspiration of the test-breakfast or test-meal. *Very little chyme* is present in these cases, but regurgitation is *in evidence.*

There are no distinctive clinical symptoms in most cases; though Knapp[1] reports diarrhea alone or alternating with constipation, as sometimes associated, and also toxemic symptoms, as does Ebstein; and Einhorn refers to 2 cases in which there was associated cardiac relaxation, with belching as the chief symptom. My prolonged investigations among the nervous and insane lead me to absolutely disagree with Knapp that achylia (functional or organic) and insufficiency of the pylorus are confounded. That author believes there is no functional achylia, and with this *I entirely disagree.*

**Treatment.**—Occasional lavage; strychnin, $\frac{1}{40}$ grain (0.00146), and belladonna extract, $\frac{1}{6}$ grain (0.011) t. i. d., with intragastric faradization (every other day).

*Massage* and *douches* are serviceable.

## SPASM OF THE PYLORUS (PYLOROSPASMUS)

Spasm of the pylorus is usually a secondary occurrence, associated with hyperchlorhydria, hypersecretion, ulcer of the pylorus or of the duodenum, or in its immediate neighborhood; and in the latter case must be considered a reflex neurosis. It may result from irritation of the stomach by indigestible diet, by food that is too hot or cold, or by strong spices, etc. It is rare with carcinoma with abundant lactic acid.

Bentijac[2] described a case who drank a glass of kerosene by mistake, and who later developed all the symptoms of stenosis of the pylorus and dilatation.

Operation showed the pylorus normal, but spasmodically contracted. Vomiting ceased after operation.

Pylorospasm may be secondary to gall-stones, gall-bladder disease, or to chronic or protective appendicitis, in which events a careful investigation of the history of the patient and the physical examination will prove of service. As a pure neurosis, spasm of the pylorus is a rare affection, and is much rarer than cardiospasm. All possible causes of spasm of the pylorus must first be excluded.

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[1] Phila. Med. Jour., May 24, 1902.

[2] Thèse de Paris, 1888.

Repeated spasm of the pylorus, in conjunction with hyperacidity or hypersecretion, is undoubtedly at times the *cause of gastric dilatation;* and in some cases *benign stenosis may result from hypertrophy at the pylorus caused by repeated spasm.*

**Symptoms.**—Pain, increased peristalsis of the stomach, and occasionally vomiting are present. The pain may radiate from the middle of the epigastrium. There may be local tenderness, and at times the pylorus may be felt to contract under the examining fingers. Retention and stagnation of food may occur and ultimately dilatation of the stomach.

Pure nervous spasm of the pylorus is difficult to diagnose. All perversions of secretion and all mechanical obstacles must be excluded as noted. Nervous pyloric spasm is most likely to be associated with the hysteric or neurasthenic condition.

The writer has seen a number of cases of pyloric spasm in which hyperchlorhydria was unquestionably the chief factor. This has been present in a number of nervous patients, especially in anemic women, and the possibility of ulcer has been suggested. Cure followed the treatment of the hyperchlorhydria. In the pure neurosis cases the gastric analysis may vary on different occasions.

**Treatment.**—Belladonna is of value to relax spasm and lessen secretion; the tincture, 10 minims (0.592) t. i. d., or extract, $\frac{1}{4}$ grain (0.016) three or four times a day. Olive oil (1 to 2 ounces) before meals lessens spasm.

The nervous system must be treated and bromids may be of service. In the secondary cases, secretory perversions and other causes of the condition must receive appropriate treatment. Lavage is necessary if there is dilatation.

Einhorn[1] has described and recommended for treatment of pyloric spasm a dilating inflatable catheter. The patient swallows a duodenal bucket the night previous, and the dilator is introduced along the thread of the bucket like the Gouley's sound along the filiform bougie. The instrument is difficult of introduction, and the method impractical and of no value. Recently Einhorn[2] has devised a small gauze balloon (pyloric dilator), to which is attached a long thin soft-rubber tube with a stop-cock and syringe at its distal end. The instrument looks much like the duodenal pump, but the action is reversed—the syringe being used for an inflator to dilate the pylorus. The balloon is swallowed in collapsed condition the night previous, and on the assumption it has passed through the pylorus,[3] the following morning dilatation of the pylorus is carried out. Einhorn reports three cures—one of spasmodic and two of organic stenosis; in the one case the stomach reaching to the symphysis, the other case was congenital stenosis. This method the writer has determined *to be unreliable*, in fact, *no cure can be effected thereby* in his belief, and in addition the suggestion that benign stenosis of the pylorus can be cured by this treatment is a *serious menace to the safety of the patient* by reason of the delay. Organic stenosis of the *pylorus is always a surgical condition.* The author has already referred to the so-called medical cures of ectasy due to stenosis of the pylorus, and how they invariably relapsed until surgical measures were undertaken.

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[1] Med. Rec., Oct. 9, 1909; Ill. Med. Jour., June, 1910; Med. Rec., June 10, 1911; Ibid, June 10, 1911.

[2] Med. Rec., Jan. 21, 1911.

[3] A diaphane is attached to the latest instrument, the light demonstrating when it has passed the pylorus (Med. Rec., June 10, 1911).

*Surgery.*—Appendectomy is indicated if appendicitis is the cause, or operation on the gall-bladder if such is at fault. Exploratory laparotomy may be necessary to determine the cause of the pylorospasm, and one may find that pyloroplasty, pylorectomy, or gastro-enterostomy may be indicated. A simple method of *closure of the pylorus* is by involution of *folds of the stomach* on the *gastric side* of the pyloric ring. Gastro-enterostomy is then performed.

## ATONY OF THE STOMACH

This is a condition in which the muscular action of the stomach is retarded and weakened, and moderate motor insufficiency results. It may be acute or chronic and secondary to other conditions, or as a primary neurosis. It has been already fully described.

## HYPANAKINESIS VENTRICULI (EINHORN)

is defined as a condition in which the mechanical function of the stomach is greatly reduced. There are no breaks, or few, in the gastrograph, an instrument to determine the churning movements of the stomach. It seems a minor degree of atony, and is of no special clinical significance.

## HYPERANAKINESIS VENTRICULI (EINHORN)

This is an excessive mechanical action of the stomach. It seems to correspond to hypermotility. Excessively active churning movements occur in this condition (Einhorn). It seems difficult to separate it from hypermotility.

## SPASM OF THE CARDIA (CARDIOSPASMUS)

Spasm of the cardia consists in a spasmodic contraction of the muscles of the cardia, with the production of pain and difficulty in swallowing (dysphagia). It may be secondary to other diseases (organic disease), reflex from local irritation, or as a primary neurosis, associated with nervous conditions.

**Etiology.**—It may be produced by the passage of the stomach-tube or after rapid eating, by very hot or cold drinks, and by coarse or hard food.

Among other causes are ulcer or carcinoma of the cardia. Hyperacidity may occasionally cause it, though pyloric spasm is more frequent with this affection. Diverticula, or inflammation of the esophagus, are causes. It may occur in tetanus. It is quite frequent in hysteric or neurasthenic individuals, especially in air swallowers (aërophagia), in whom spasm of the pylorus may also occur with resulting distention of the stomach (" pneumatosis "). Violent psychic shocks may also produce it, so the condition may be a primary neurosis. Fermentative processes in the stomach, with the production of gas, may be a cause; but organic disease is usually associated. *Primary spasm of the cardia cannot produce gastric distention* unless spasm of the pylorus is also present.

**Symptoms.**—*As a pure neurosis* the attack usually begins suddenly. The patient may be in perfect health, when an acute pain begins in the region of the cardia radiating toward the chest or back. It occurs generally during the ingestion of food, and there is a feeling as if some of it were arrested. There is at times some interference with breathing. Slight dyspnea is present and the respiratory movements are more forcible. There may be regurgitation and the patient feels better thereafter. There is gagging and often vomiting.

The attack can be acute and last only a short time, or the condition may become chronic. The dysphagia in these cases, as a rule, increases; and though for a time the patient, by taking a deep inspiration and compressing the thorax while holding his breath, can force down the food, the deglutition of solid food becomes more and more difficult, and finally, only liquids can be taken. Emaciation may become quite marked, and after several *years atonic dilatation of the esophagus* may result similar in character to atonic gastric dilatation from pyloric spasm. Dilatation of the esophagus may also be produced by benign or malignant stenosis at the cardia; or by paralysis of the dilator nerves of the cardia, from paralysis of the esophagus, or by loss of *reflex relaxation* of the cardia (Einhorn).

**Diagnosis.**—In acute cases the existence of pain and dysphagia, the delay or absence of the swallowing sound, and the spasmodic resistance at the cardia on insertion of the stomach-tube, which can be overcome by pressure, together with the presence of neurasthenia or hysteria, settle the diagnosis.

In the chronic type the history of the case, the fact that often the resistance to the *large tube* is less than to *the small one in nervous cases*, all aid the diagnosis. Dilatation of the esophagus may finally result.

In true stenosis (organic stricture) the small sounds are more readily passed. The method of determining esophageal stricture has been described. If blood appears with the sound, ulceration is at once suggested. Cachexia is suggestive of malignancy, and aspiration of the gastric contents will settle the question. Dilatation of the esophagus occurring as a result of cardiospasm can be further determined as follows:

A slice of bread can be dissolved in 200 c.c. of water to form a soft mass, and administered, the patient being instructed to employ forcing movements, so that it will enter the stomach. An hour later 200 c.c. of water may be given, no forcing movements to be employed. In about three to five minutes an aspirating tube can be passed to the cardia, and if dilatation and spasm exist the water just ingested can be removed, and will be in the same condition as taken. The tube should then be forced on into the stomach and the true gastric contents removed. These are tested. The administration of bismuth with the employment of the *x*-rays is also of value for the purpose of diagnosis of dilatation of the esophagus. Plummer's methods for determination of stenosis of the esophagus are described under that subject.

**Prognosis** is good in the acute cases, but as regards complete cure in chronic cases it is not so good, though there is no danger to life, as a rule, except in cases with marked dilatation of the esophagus.

**Treatment.**—If the spasm occurs while eating and drinking. they should be performed slowly. The food should be well masticated or should be, preferably, soft in character; and excessively hot or cold food, rich, spiced food, etc., should be avoided. Nutritive enemata to rest the esophagus may temporarily be required.

*General tonics should be given.* The employment of sounds, left in place for some time, is of value.

Tincture of belladonna, 10 minims (0.592), should be given t. i. d. to relieve spasm. Anesthesin, 5 grains (0.3) t. i. d., relieves pain. The bromids are of service; thus, sodium bromid, 15 to 30 grains (1.0–2.0) t. i. d.

Opium or chloral should be administered with caution, or not at all.

In the chronic cases soft or liquid food should be given. The patient should bear down to force the food thoroughly by the obstruction. Olive oil, 1 to 4 drams t. i. d. before food, is of value. The stomach-tube should be introduced at least once a day to relax the cardia; if there is emaciation, food can be administered through the tube; if the esophagus is dilated, it can be emptied and washed out. Dilatation of the cardia may be necessary. There are numerous inflatable bags and metal dilators (Einhorn's,[1] for example) devised for this purpose. For severe cases the writer prefers Plummer's method with olives, or a stomach-tube with the thread guide, as already described. He also has an inflatable rubber dilator, which can be guided by the same method. Rectal feeding must be enjoined for a time in severe cases. As improvement occurs the diet can be increased.

[1] Amer. Jour. Med. Sci., Oct., 1910.

## INSUFFICIENCY OF THE CARDIA

Under this we may classify *eructation, singultus, pyrosis, regurgitation,* and *rumination.*

**Eructation (Belching).**—Eructation may be defined as the expulsion of gas from the stomach through the mouth.

The condition may be secondary to various conditions of the stomach or may be a *primary neurosis.*

Normal healthy subjects may eructate after drinking carbonated water, or those who eat rapidly without proper mastication, and thus swallow considerable air.

*Secondary Cases.*—Belching may be associated with acute or chronic gastritis or with fermentative processes in the stomach.

The clinical symptoms, gastric analysis, and fermentation test will render a positive diagnosis.

In fermentative processes the gas is often *malodorous.*

*Primary Cases.—Nervous Belching (Eructatio Nervosa).*—We have cases in which belching occurs at short intervals, which is independent of the character of the food and clearly of nervous origin. It is noisy, as a rule, and may persist for a considerable period or occur in paroxysms.

The gas consists of atmospheric air which has been swallowed (aërophagia) or aspirated into the stomach.

Osler claims that the stomach acts as an elastic sac, and can aspirate the air without the effort at swallowing, and that it tends to fill after expulsion. Some of the air probably comes from the esophagus and has been swallowed just previous to the act of belching. Bouveret considers it due to *spasm of the pharynx.*

**Etiology.**—This nervous type of eructation is observed in hysteric women and chiefly in neurasthenics, or after excitement, shock, or mental worry. It frequently occurs in children, often in several of a family.

The belching may last for a long period, only occur during the day, and may be extremely annoying.

**Diagnosis.**—Absence of fermentation, as shown by the test, the history of nervousness or of shock, and the gastric analysis showing normal secretion render the diagnosis conclusive.

**Treatment.**—Massage, hydrotherapy, iron and arsenic, change of climate, the faradic current (extraventricular or intragastric), bromids, and suggestion to the patient are of service.

## SINGULTUS GASTRICUS NERVOSUS

Singultus or hiccough may last for a few minutes, several hours, or even for some days. It may cease and then recur. Hiccough has even resulted in a fatal issue, the patient dying of exhaustion.

**Etiology.**—Among the various causes are diseases of the viscera, such as gastritis, hyperchlorhydria, atony of the stomach, acute or chronic ectasy, carcinoma of the stomach, catarrh of the intestines, intestinal obstruction, peritonitis, appendicitis, intestinal paresis, pancreatitis, diseases of the liver, typhoid fever, pulmonary disease, *alcoholism*, anesthesia, diabetes, gout, nephritis; diseases of the nervous system, such as epilepsy, meningitis, brain tumor, etc. It *may frequently* occur *with hysteria.*

As *a pure neurosis* it is an *extremely rare condition*, though I agree with Bassler[1] that it may occur, though associated with hyperesthesia of the stomach or with gastralgia. It must be diagnosed by the process of exclusion and I believe most of the so-called cases of singultus gastricus nervosus are really a symptom of the hysterical condition.

**Treatment.**—The *gastric functions* must be examined in all cases and *functional disturbances corrected.* Iron, arsenic, sodium bromid, strychnin, and, at times, belladonna are indicated. The general nervous condition of the patient must be treated and the physical condition improved. During a severe attack inhalation of amyl nitrite will at times be beneficial.

### PYROSIS (HEART-BURN)

This consists in the ejection of chyme from the stomach into the esophagus, with which is associated a burning sensation in the epigastrium.

**Etiology.**—It most frequently occurs with hyperchlorhydria, but has been found with achylia.

It may occur as a neurosis, especially among the hysteric or neurasthenic, with normal gastric contents. It is of mixed type, motor and sensory.

General tonics and electricity are of value in these cases.

### REGURGITATION

Regurgitation denotes the condition in which, after eating, some of the food ingested, liquid or liquid and solid, rises from the stomach, enters the mouth, and is ejected.

Occasionally, small fragments are swallowed again. The act is usually involuntary, though it can be produced by some at will. Early regurgitation tastes of the food, later it is acid. The patients *can suppress it at will*, and in this way it differs from regurgitation due to cardiac stenosis or diverticula.

The passage of the esophageal sound or olive and chemic analysis also give aid in diagnosis. In some cases, if regurgitation is marked, considerable emaciation may result.

Organic disease, such as stricture of the esophagus, must be excluded; also gastritis.

**Etiology.**—Patients of nervous or hysteric type develop this condition; and mental worry and nervous strain may be the cause.

**Prognosis** is good.

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[1] N. Y. Med. Jour., Aug. 13, 1910.

**Treatment.**—The patient should eat slowly and masticate his food. Suggestion is good; also forbid the patient to spit out his food, and tell him to swallow it again.

Massage, faradization (intragastric and extragastric); strychnin, $\frac{1}{40}$ grain (0.0016) t. i. d., and the treatment of the nervous condition are indicated.

## RUMINATION

(*Synonym.*—Merycism, "Chewing the cud.")

In this condition the food is returned from the stomach, without nausea, into the mouth, some time after meals, where it is chewed and swallowed again.

**Etiology.**—It occurs more frequently in men than in women; and most of the cases reported belong to the higher classes, especially among professional men.

Many of the lower class would not deem the condition abnormal, and women would conceal it, so it may be said to belong to all classes. It occurs among hysteric persons, neurasthenics, epileptics, and idiots.

Possibly heredity and, undoubtedly, *imitation* in many cases are factors. Occasionally there may be a pathologic condition of the stomach, with regurgitation, and rumination follows. Several cases have been reported of imitation among children.

Shock, trauma, rapid eating, and emotional disturbances in nervous persons are at times accessory factors.

*Gastric Findings.*—In some cases hyperacidity has been found, and rumination diminished after this was corrected.

Diminished acidity has been found by Boas, and improvement followed after hydrochloric acid was administered.

On the other hand, achylia gastrica has been present in such cases and also normal secretion.

The *motor functions* are normal in most cases.

**Prognosis.**—The habit may be corrected in part, but may persist for years, with no impairment of health. In some patients the attacks are periodic.

**Treatment.**—Any secretory anomaly that exists must be corrected. The treatment should be by mental impression (psychic) on the part of the physician. The patient should be taught that he can readily suppress the condition. Small bits of ice after meals, lavage, and gavage have been suggested. The nervousness should be corrected by iron, strychnin, and arsenic.

## NERVOUS VOMITING (VOMITUS NERVOSA)

Vomiting is a complex process. There is a contraction of the abdominal muscles and fixation of the diaphragm, with a contraction of the stomach, accompanied by closure of the pylorus and opening of the cardia; reversed peristalsis of the esophagus and closure of the larynx and posterior nares occur, with resulting expulsion of the stomach-contents through the mouth. The vomiting center lies in the medulla near the vagus center.

Vomiting may be due to some organic disease of the stomach or as a result of irritation due to the ingestion of food of abnormal character. Certain intoxications, as from uremia, cholemia, opium, morphin, tobacco, ether, or chloroform, may produce vomiting.

Nervous vomiting is characterized by the absence of these conditions just mentioned, and may be either cerebral or spinal in origin, or due to reflex disturbance, overexertion, emotional shock, anger, fright, or neurasthenia.

Among the cerebral causes are organic disease of the brain and its meninges, concussion, and tumors.

Spinal forms, such as tabes, or occasionally paresis, multiple sclerosis, and subacute myelitis.

Reflex causes, such as from the pharynx, larynx, palate, liver, peritoneum, kidneys, genitals, etc. Juvenile vomiting and the periodic or cyclic vomiting (Leyden), also the cyclic vomiting of children (infants) are included.

**Characteristics of Nervous Vomiting.**—Stiller calls attention to the following peculiarities:

This type of vomiting seems *to be independent of the character and quality of the food, and occurs generally without any premonitory symptoms.* Sometimes digestible foods are vomited, while indigestible material is well borne. There seems at times to be a power of selection to vomit certain nutriment, and the patients suffer from no inanition. Vomiting may also occur from the empty stomach, and the condition is associated with various nervous symptoms.

Boas has noted that the motor and secretory functions are normal, though Bouveret and Einhorn have observed a diminution or absence of secretory function in some cases.

**Treatment.**—Tonics. Correct disturbance of secretory functions. Mental impression on the part of the physician is important. Change of scene is at times of value.

## PERIODIC VOMITING (VON LEYDEN)

There are several particulars connected with this type of vomiting. As a rule, *no cause can be discovered,* and the attack appears when the patient is in perfect health. The attacks occur at intervals of equal duration. After the attack the patient is immediately restored to health, and remains well until the next one occurs.

They resemble somewhat the gastric crises of tabes, and are similar in suddenness of occurrence to violent attacks of migraine. They seem to be influenced by emotional disturbances. They begin with slight nausea and with a chilly feeling and headache, followed by vomiting of the gastric contents, and, later, bile and mucus streaked *with blood.* Lerche[1] reports a case in which the patient vomited from a few tablespoons to a cupful of blood during each attack.

The vomiting is very persistent, and all food or even small quantities of water are ejected. In some cases there is severe pain. The patient becomes greatly prostrated, the abdomen sunken, and the face pale. The attack may last from a day to a week or longer. Suddenly the nausea and vomiting cease, the appetite returns, and the patient is rapidly restored to health.

**Gastric Juice.**—This has been found *to be normal in most cases,* though Einhorn reports a case of achylia gastrica. On the other hand, with gastrosuccorrhea, the content of hydrochloric acid is high, a valuable aid to differential diagnosis.

**Treatment.**—Rest, ice pellets, morphin, $\frac{1}{4}$ grain (0.016), by hypodermic, and belladonna tincture, 10 minims (0.59), are useful. Cocain I deprecate. Tincture of hyoscyamus, 30 drops (2 c.c.), and hot applications during attacks. Oxalate of cerium in grain doses is of value. Between attacks, tonics, change of climate, and hydrotherapy. Avoid opiates as far as possible.

### Cyclic Vomiting in Children

This is probably due to faulty metabolism; occurs generally in those from two to four years of age, of a nervous type and gouty family history.

Griffith[2] holds that cyclic vomiting may give symptoms very similar to appendicitis, the absence of signs of local inflammation being the chief differential point.

Russell[3] reports a case of cyclic vomiting which autopsy apparently showed was due to *recurrent spasm* in an hypertrophied and somewhat stenosed pylorus.

There are premonitory symptoms; temperature but seldom over 100.5° F., anorexia, languor, followed by persistent and violent vomiting of food, mucus streaked with blood, and at times bile. Prostration is marked; the gastric contents very acid. Acetone, diacetic acid, and oxybutyric acid are present in the urine, and so it is believed it is a form *of acid intoxication;* others consider the acidosis a result of the condition; uric acid in the urine is diminished. It is possibly a lithemic manifestation.

The attack resembles migraine in the adult. Antecedents are a neurotic and gouty family history. Carbohydrates in excess seem to have a bearing on its production, according to some; fatigue, excitement, or tonsillitis bring it on.

Comby[4] believes that cyclic vomiting is generally due to chronic appendicitis and that cures have followed in many cases as a result of appendectomy.

**Prognosis.**—Good.

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[1] Med. Rec., April 30, 1910. [2] Amer. Jour. Med. Sci., cxx, p. 553.

[3] Brit. Jour. of Children's Diseases, Feb., 1910.

[4] Archiv. f. Kinderheilkunde, vol. l, 1909, p. 33.

**Diagnosis.**—Attacks are not brought on by indigestible food; the disease is self-limited, and the child rapidly returns to the normal state. The attacks are repeated.

**Treatment.**—Calomel to abort it; food and drink should be stopped; enemata and enteroclysis are advisable. Hypodermoclysis may be necessary in severe cases and rectal feeding is indicated.

*Alkaline Treatment.*—Sodium bicarbonate, 15- to 30-grain (1.0–2.0) doses, may be given t. i. d. by mouth, or double dose by rectum if necessary.

Limit starchy food. Care must be taken not to tire the child thereafter.

### JUVENILE VOMITING

Overwork at school is often the cause. Cardialgia and vomiting occur.

There may be headache, slow pulse, pallor, dilatation of pupils, etc.

*Treatment.*—Tonics, proper diet, removal from school.

### REFLEX VOMITING

Nervous vomiting is frequently reflex in character from disease of almost any organ. Among the causes are disease of the pharynx, elongated uvula, diseases of the abdominal organs, as hydronephrosis, movable kidney, kidney colic, ptosis of the liver or spleen, peritonitis, appendicitis, hernia, and disease of the sexual organs. These conditions should receive appropriate treatment.

The vomiting of pregnancy belongs to this type.

Belladonna tincture, 10 minims (0.59), t. i. d.; cerium oxalate, 2 grains (0.13), t. i. d.; Fowler's solution of arsenic, several doses 1 drop each; bromids, 15 grains (1.0); codein, ¼ grain (0.016); or chloral, 3 grains (0.194), t. i. d.

| | | |
|---|---|---|
| ℞. | Menthol | gr. x (0.6); |
| | Syrup | ℥ij (60.0); |
| | Aq. destil. | q. s. ℥iv (125.0).—M. |

Sig.—Two teaspoonfuls t. i. d.

The use of cocain I deprecate. Lavage is temporarily of service.

Abortion may occasionally be required in pregnancy cases. Change of scene may be necessary.

### IDIOPATHIC NERVOUS VOMITING

In some hysteric or neurasthenic persons (adults) vomiting will occur after meals without any apparent cause, more frequently in women and without showing the periodic type. Usually part of the meal is vomited. It may continue for a long period. Nutrition is frequently not disturbed. The vomiting may occur so quickly during the act of ingestion of food that it seems as if it did not enter the stomach, but was rejected by the esophagus. Other patients seem normal in this regard.

Occasionally hysteric subjects may vomit blood. In all cases the gastric secretion must be studied, so as to exclude hyperchlorhydria, hypersecretion, etc., and to observe whether vomiting complicates some other disease or is a reflex affection.

**Treatment.**—Suggestion, the regulation of the mode of life, tonics, such as arsenic and iron, bromids; change of climate; gavage for about two weeks; lavage, with 1:1000 nitrate of silver, has proved of value.

Faradization, extra-abdominal or intragastric (Einhorn), has been of benefit.

### PNEUMATOSIS

Spasm of the cardia, combined with pyloric spasm, may produce pneumatosis (distention of the stomach with air), with a resulting sensation of tension and at times dyspeptic asthma.

Hysteric or neurasthenic symptoms are associated.

Aërophagia (air swallowing) is probably the cause. The upper part of the abdomen is markedly distended and tympanitic, and there is interference with respiration. As a rule, there is no belching.

**Diagnosis.**—Organic affections of the stomach must be excluded, in which the gas has a foul odor and the contents ferment.

**Treatment.**—Tonics and bromids are indicated. Aspiration of the stomach in the acute attack, so as to give exit to the air, is the best method. It may be necessary to repeat it.

Extract physostigmatis, $\frac{1}{8}$ grain (0.008), or eserin, $\frac{1}{100}$ grain (0.00065), or morphin, $\frac{1}{4}$ grain (0.016), or tincture of belladonna, 10 minims (0.59), or extract of belladonna, $\frac{1}{4}$ grain (0.016), may be required. A little peppermint water or spirits of chloroform aid belching of gas.

### SECRETORY NEUROSES

The secretory function of the stomach is undoubtedly under the direct control of the nervous system. For example, in a hungry dog with a fistula the sight of meat will produce gastric secretion, and in the case of a man with impermeable esophagus, but with a gastric fistula, mastication produced gastric secretion. The vagus has been demonstrated to be the secretory nerve. The stomach itself, however, possesses some secretory power, since after section of the *pneumogastric and sympathetic nerves*, secretion will occur after the application of an irritant.

Hyperchlorhydria, gastrosuccorrhea (hypersecretion), and achylia gastrica may all be functional disorders of secretion, and have been described in special chapters.

Subacidity (hyposecretion, hypochylia gastrica, hypochlorhydria) may be of nervous origin, and must be differentiated from cases occurring with organic disease of the stomach, especially gastric catarrh.

Hydrochloric acid, strychnin, massage, and electricity are of service in these last cases.

In the subacid cases sudden changes in the gastric findings (secretory) are in favor of a neurosis, according to Hemmeter.

Disorders of secretion may accompany other diseases, such as tabes or spinal lesions.

## NERVOUS DYSPEPSIA (LEUBE)

(*Synonym.*—Neurasthenia Gastrica.)

Leube originally described nervous dyspepsia as a disorder of the stomach, characterized by a variety of distressing subjective symptoms during the act of digestion, but in which it was *normal as regards time and chemism.* In effect, he originally considered "nervous dyspepsia" as a neurosis of sensibility. He has more recently extended his definition to other forms. Strictly speaking, nervous dyspepsia is a combined *gastric neurosis* in which the sensory disturbances (subjective symptoms) are the most prominent.

It may be combined with secretory and, at times, even with motor disturbances.

**Gastric Juice.**—The findings in the gastric juice are not characteristic. It may be frequently normal; it may occasionally be hyperacid, *more frequently diminished acidity*, or at times there may be variations in the same subject. In long persistent cases atony may be present.

Some authors refer to the presence of enteroptosis or membranous colitis with neurasthenia gastrica. In this connection it is evident that frequently the diagnosis of "nervous dyspepsia" is made, when, in reality, gastroptosis (enteroptosis) is the basis of the difficulty. With the ptosis of the viscera we have sensory, secretory, and frequently motor disturbances of the stomach, associated with nervous symptoms, but the correction of the ptosis will cure the condition.

In pure nervous dyspepsia *all organic lesions of the stomach* must be excluded, and also *ptosis of the viscera.* The stomach *must occupy the normal position.*

**Etiology.**—Neurasthenia gastrica may occasionally appear as an independent neurosis, but more usually with nervous symptoms, hysteria, or neurasthenia. Grip, pulmonary disease, anemia, chlorosis, malaria, or debilitating conditions predispose to it, as do also reflex irritation from the sexual organs, excessive venery, abuse of alcohol, or tobacco.

*Age and Sex.*—This disease occurs more frequently in men than in women, especially among brokers and those subject to worry and mental strain, and usually at the prime of life, between thirty and fifty, though it may be present at other periods.

**Symptoms.**—The patient generally complains of a feeling of fulness or pressure after eating, or even of slight pain or belching, loss or irregularity of appetite, a sleepy feeling, or even weakness or dizziness. The tongue is usually clean. There is marked mental depression, and the patients are nervous and anxious. One *peculiarity is that the quantity and quality* of the food *seem to make little difference* in their *symptoms.* At times indigestible food can be taken without discomfort, while at other times digestible food may produce the symptoms. Occasionally the pain and discomfort are present when the stomach is empty. There is often nausea and occasionally vomiting. Thirst is variable. There is usually tension or fulness of the intestines due to accumulation of gas, which is passed later per rectum. The bowels, as a rule, are constipated, with occasionally an alternating diarrhea. The movements may appear in narrow cylindroids or small balls in some cases.

In addition to the feelings of depression, insomnia, palpitation, headache, and lassitude may be present; also vertigo, impotence, or emissions.

The surroundings and general mood of the patient have a marked influence on the symptoms. The cases are often extremely disagreeable in their "home life." Circumscribed points, sensitive to pressure, have been described as diagnostic; one below the ensiform and the others near the navel, especially to the left, but Ewald has demonstrated "nervous dyspepsia" in which no such points could be found.

**Course.**—The course of the disease is slow and the symptoms vary; sometimes one symptom being prominent; at another time, another. The mood of the patient has a marked influence, and in good company he may forget his trouble. They generally do not suffer much in nutrition, but in cases with insomnia they at times lose considerable weight.

**Diagnosis.**—The presence of general nervous symptoms, especially of those pointing to the stomach, *without the presence of organic disease of the organ;* the fact that the gastric secretion is often found to be normal, though at times hypochlorhydria and more rarely a mild hyperchlorhydria, and that we frequently obtain variable gastric analyses in the same patient; that there is lack of proportion between the gastric findings and the condition of the digestive organs, as compared with the severe complaints voiced by the patient; that the character of the food, whether digestible or indigestible, seems to make no difference as regards increasing or ameliorating the symptoms, and, finally, that change of scene or the mental condition of the patient has a decided influence on the condition—all these facts point to neurasthenia gastrica. The writer believes pure nervous dyspepsia to be rather rare, and that it is often thus incorrectly diagnosed.

**Differential Diagnosis.**—The chief differences are as follows:

*Neurasthenia Gastrica.*—Character of the food makes no difference as to symptoms. There are sudden changes in the patient's condition: well for a few days, and then marked symptoms; nervous symptoms marked. Gastric secretion often normal, and frequently variable in the same patient at different times.

*Chronic Gastritis.*—Aggravated by errors in diet; symptoms constant; *mucus in the gastric contents;* reduction in hydrochloric acid is marked as a rule.

*Ulcer of the Stomach.*—Painful area in the epigastrium, tender on pressure; hematemesis or occult blood, melena, and pain is increased markedly after ingestion of food. The character of the food influences the pain markedly, even in the more obscure cases. Pain remits or disappears.

*Cancer.*—Age of patient; tumor; cachexia; quality and quantity of the food may not markedly influence the pain; gastric analysis usually showing absence of free HCl, lactic acid, and Boas-Oppler bacilli present; pain continuous; progressive emaciation.

**Treatment.**—If there are any sexual disorders which reflexly might affect the nervous condition, these should be treated; sexual excesses or overindulgence in alcohol or tobacco should be checked. Brokers and professional men who have mentally overexerted themselves or are tired with the worry of business cares, lead an irregular life, or who are engaged too actively in social pursuits, if possible, should have a change of scene. Horseback riding, golf, yachting, fishing, shooting, camp life for a few weeks, a pleasure trip, all give excellent results. A short ocean trip south or abroad is of service. For those who cannot afford these methods, the lightening of business and professional cares is important.

Hydrotherapy, massage, electricity, especially by the faradic current, combined with out-of-door life and proper exercise, mild gymnastics, so as not to tire the patient, are of value. *The mental impression* created by the physician *is important.* Static electricity is at times of service. General faradization, the bare feet on one electrode, and the other being passed over the body, is useful (Rockwell).

The diet should be abundant, the patient avoiding highly seasoned food, alcohol, strong coffee, and excessive smoking.

Stomachics should be given if hydrochloric acid is deficient, such as nux vomica, compound tincture of cinchona, dilute hydrochloric acid, etc.

If hyperchlorhydria, then the alkalis should be administered. The patient should secure the proper amount of sleep.

Forced feeding and the Weir-Mitchell rest cure are necessary in severe cases. Milk and raw eggs are of value in reduced nutrition.

Malbranc's gastric douche has been recommended in some cases, and Einhorn suggests the use of his gastric spray. Personally I do not employ such local measures.

The general tone of the patient should be built up by iron, such as peptomangan (Gude's), iron pills, iron tropon, arsenic, and strychnin. Small doses of nux vomica, combined with compound tincture of cinchona, are excellent to improve the appetite:

℞. Tr. nucis vomicæ........................ ʒiij (12.0);
Comp. tinct. cinchona...................... ℥ss (16.0);
Aq. destil............................q. s. ℥iv (125.0).—M.
Sig.—1 to 2 teaspoonfuls t. i. d. in water before meals.

Basic orexin, 3 grains (2 decigrams) t. i. d., has also been recommended by Einhorn for this purpose.

Sodium or ammonium bromid, 5 to 10 grains (0.3–0.6) two or three times a day, lessens the nervousness.

The bowels should be properly regulated by cascara, aloin pills, phenolax, etc. Iron springs, such as Franzensbad, or salines, as Kissengen or Weisbaden, are of service.

The carbonated bath (Nauheim, Triton) I have found—given every other day at home for a course of 12 baths at a temperature of 95° to 98° F.—to be of service in toning up the circulation and general nervous condition.

## CHAPTER XVIII

# DYSPEPTIC ASTHMA

This type of asthma, due to digestive disturbances, was first described by Henoch,[1] later by Silberman,[2] Oppler,[3] Boas,[4] Murdoch,[5] and Einhorn.[6] Many others have described this condition.

The symptoms first reported by Henoch were of an acute type, and, according to his belief, were the result of reflex action starting from the stomach. It is noteworthy that the *most severe symptom disappeared when the patient vomited.* In his cases the breathing was rapid and shallow, pulse rapid and feeble, *and at times so rapid* that it could not be counted, extremities cool, the temperature subnormal, and there were even symptoms of collapse.

In all cases there was acute dyspepsia due to some error in diet. The region of the stomach was usually distended and painful. The greatest number of cases were observed in children. Numerous explanations have been given for this condition, and a variety of experiments have been performed. Henoch believes it to be due to reflex action, starting from the stomach and causing a vasomotor spasm; while Einhorn suggests reflex irritation of the vagus fibers. In view of the fact that the majority of cases occur after dietetic error, Riegel's suggestion of auto-intoxication as a factor seems to have a *decided bearing* on the subject.

On the other hand, under Acute Dilatation of the Stomach I referred to certain peculiar clinical types, in one of which many of the symptoms resembled angina pectoris, there being dyspnea and rapid and feeble heart action, and in some attacks loss of consciousness. These attacks followed indiscretion in diet. In view of this fact and also that Henoch describes distention of the stomach as present in most cases, and dietary indiscretion as a cause, it would seem to me that in this type at least acute gastric dilatation from auto-intoxication is a factor. Einhorn describes acute cases following excesses in eating, drinking or smoking, excitement, or from no discoverable cause. Even these facts do not mitigate against the theory of distention.

The second group which he classifies are more of a chronic type, appearing after meals, or after overexertion, or without apparent cause, or those appearing several hours after meals spontaneously, or after exertion.

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[1] Berlin. klin. Wochens., 1876, No. 181.
[2] Ibid., 1882, No. 23.
[3] Allgem. Med. Central. Zeit., 1890, No. 71, p. 849.
[4] Arch. f. Verdauungskrank., Bd. 11, 1896, p. 444.
[5] N. Y. Med. Jour., Jan. 12, 1901.
[6] Jour. Amer. Med. Assoc., Feb. 1, 1902.

In some of these cases a small amount of food would check the attack. The last type would seem to suggest reflex irritation from hyperchlorhydria, for example, which when relieved would stop the asthma.

Some of the last group present, in some cases, symptoms suggestive of pseudo-angina pectoris.

*Secretory Functions.*—Achylia gastrica has been found in some patients and hyperchlorhydria in others. Treatment of these conditions caused subsequent disappearance of the attacks of asthma. With achylia the coarse particles of food, Einhorn believes, might cause reflex irritation of the vagus. Asthma dyspepticum may also occur with chronic gastritis.

On the other hand, these attacks have occurred in patients in whom the gastric secretion was normal; and Boas believes that hyperesthesia of the stomach with reflex irritation is the cause.

Again, in my case of acute dilatation with some of the attacks similar to angina, the gastric secretion was normal; but dietary indiscretion produced gastric distention and the attack noted. It was immediately relieved by vomiting.

*Floating liver* was noted by Einhorn in 5 cases, and this together with abdominal ptosis of other organs; and he believes that the prolapsed liver, by dragging down the diaphragm, may be a cause of this type of asthma. In my own opinion ptoses of the viscera have an influence on the secretory conditions in the gastro-intestinal tract, and only to this degree predispose to dyspeptic asthma. Probably these various factors to which I have referred may have a bearing in different cases.

**Treatment.**—Disorders of the gastric secretion must be appropriately corrected, and ptosis of the viscera supported by Rose's adhesive belt.

Excesses in the use of alcohol and tobacco must be corrected, mental worry and strain be avoided, and the mode of life must be regulated.

All indigestible food must be avoided and diet suitable to each case must be instituted. By this means many cases will be relieved and often cured.

In acute cases with distention of the stomach, lavage is indicated, also calomel, 5 grains (0.3), followed by a saline cathartic. Catharsis is also indicated in cases following excesses in eating or drinking.

## CHAPTER XIX

# THE STOMACH FUNCTIONS IN DISEASES OF OTHER ORGANS

UNQUESTIONABLY there are few diseases, either constitutional or local, which are not attended to a greater or lesser degree by some disturbance of the digestive organs. These are dependent on the general disturbance of the organism and are appropriately discussed under the symptoms of each disease.

In the present chapter I shall only briefly refer to those diseases in which disturbances of the gastric functions are particularly conspicuous.

### FUNCTIONS OF THE STOMACH IN ACUTE FEBRILE DISEASES

Numerous investigations have been carried out both on animals and men, in some of which at least accurate quantitative gastric analyses were performed. Riegel concludes that we are probably justified in stating that in acute febrile infectious diseases the production of hydrochloric acid was more frequently reduced than normal, and the secretion of pepsin is, as a rule, unchanged. Probably the fever is responsible and the condition is temporary. Von Noorden[1] showed that hydrochloric acid reaction can be obtained in fever cases if pepper and salt are administered with the food.

Some interesting researches have been carried out in numerous cases of typhoid and pneumonia. During the high temperature of these diseases there was a marked diminution, and in some cases an absence of hydrochloric acid. During defervescence an increase of the secretion was noted. During high temperatures pure milk was found in the stomach in a curdled condition several hours after the normal time for evacuation, and in one case, on autopsy, the stomach was found filled with the curdled milk of previous feedings, thus demonstrating a *diminution of the motor function.* Water alone was then administered at this period. During lower temperatures the administration of foods freely soluble in water, such as broths and gruels, were found to be best. There were less fermentation and distention under this method of feeding and the stomach more readily emptied itself.

In my own experience[2] I can confirm the fact that during the high temperature of typhoid the free hydrochloric acid diminished, often markedly, and the *motor function is not as active.* It has also been found that biliary secretion is disturbed during high fever. Stolmkow noted disturbances in the pancreatic secretion during high temperature. These facts demonstrate that the excessively high calorie food values advocated by some in typhoid fever are unscientific.

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[1] Lehrbuch der Pathologie des Stoffwechsels, 1893.

[2] Amer. Med., May, 1909.

## CHRONIC FEBRILE CONDITIONS

Observers vary considerably in their findings in the gastric secretion in these cases, some noting no changes whatever. In my own experience the temperature, as a rule, caused disturbance in the secretory function of the stomach, lessening the HCl production. The type of disease, the physical condition, the personal equation of the patient, and, most important, *the time at which the analysis is made, all have a bearing.* I shall refer to the work of Hildebrandt on this subject, under Tuberculosis. The power of absorption seems to be impaired in fever (Sticker).[1]

## CONDITION OF THE STOMACH IN PULMONARY TUBERCULOSIS

Phthisis quite frequently begins with dietetic disturbances, such as heart-burn, belching, pressure, nausea, loss of appetite, constipation alternating with diarrhea, and even vomiting, with the lung symptoms so slight as to be at first overlooked. It has been called pretubercular dyspepsia. In the later stages we may have the dyspeptic symptoms quite marked.

Hildebrandt[2] found the following results: The cases in which free *hydrochloric acid* was present usually had no fever, while those in whom it was absent suffered from continuous fever. When it was absent at one part of the day, it was when the temperature was high; and when present at another part, it was when the temperature was low. The *temperature, therefore, exercised an influence.* These findings were in advanced cases. Klemperer[3] studied 10 cases in the initial stage, 3 in the advanced, and 1 in the moderate.

In the beginning the secretory power of the stomach was usually increased, often normal, and rarely reduced. In the terminal stages, always greatly reduced. Motor reduction slight in the initial stages, reduced in later stages.

Brieger[4] analyzed 64 cases: 31 advanced, with continuous fever; 37 moderate, with more or less fever; 6 incipient cases, with no fever. Gastric secretion normal in 16 per cent. of advanced cases, and in the others insufficiency of varying degrees; in 9.6 per cent. absence of free hydrochloric acid. In moderate cases, 33⅓ per cent. normal, and in all others perversion; and in 6.6 per cent. normal secretion absent. In the initial cases, normal secretion and perversion were equally divided.

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[1] Deutsch. med. Wochens., 1889, No. 15.
[2] Berlin. klin. Wochens., 1885.
[3] Ibid., 1889, No. 2.
[4] Deutsch. med. Wochens., 1889, No. 14.

Einhorn[1] has shown in analyses of 15 cases that the appetite does not seem to correspond, as would be expected, to the gastric findings; and also that frequently the subjective symptoms do not harmonize with the objective findings.

**Treatment.**—The main indication is to improve the resisting power of the patient against the primary disease.

*Forced feeding,* especially by Russell's method, rest in bed, and fresh air, with milk and vegetable juices, I believe the best treatment for the tuberculosis. I have seen a gain of 15 to 25 pounds in each case by his methods at Ward's Island in 12 cases in eight weeks.

The heart and kidneys must functionate properly for success with his method. *Functional disturbances of the stomach* should be treated. Tuberculous ulcer of the stomach is occasionally met with in connection with tuberculosis of the other organs; the primary condition is rare.

## CHLOROSIS AND ANEMIA

Among the gastric symptoms in these conditions are found gastralgia, anorexia, hyperesthesia of the stomach, hyperchlorhydria, and chronic atony.

These symptoms appear more frequently after eating than on an empty stomach, and occur, as a rule, in attacks at irregular intervals. There are often perversions of appetite. The atony, if neglected, may progress to chronic atonic ectasia.

In chlorosis (primary anemia) the hydrochloric acid secretion is, as a rule, *increased.*

In anemia (secondary), on the other hand, depending on the causative disease we may find variable results; hydrochloric acid decreased, normal, or (more rarely) increased.

In this I agree with Riegel.

The relation between achylia gastrica and pernicious anemia has already been described. The relation of intestinal putrefaction to this disease is described later.

Many of the derangements belong to the neuroses and are dependent on the condition of the blood. The administration of iron is *chiefly indicated,* with the additional correction of the functional disturbance, if such be present.

## HEART LESIONS

In general we may say that while compensation is present, in many cases the stomach functions are normal or nearly so; with imperfect or failing compensation, with resulting stasis and hyperemia in the gastric mucous membrane, I have noted diminution in the amount of free hydrochloric acid with accompanying digestive disturbances, belching, pressure, anorexia, and at times nausea and even vomiting, with sick headache. These conditions improved after *treatment was directed to the circulation.* In severe cases free HCl may be absent. In one case with poor compensation and frequent gastric attacks, a course of treatment at Nauheim produced excellent results.

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[1] Loc. cit.

Symptoms simulating heart lesions may be produced by gastric disorders, thus: Ulcer, chronic ectasy, and chronic gastritis may produce bradycardia or arhythmia; or tachycardia may occur with chronic gastritis, in nervous gastric disorders, or with atony.

Tachycardia with acute dilatation of the stomach, especially with existing heart lesions, the author has described in the chapter on Acute Ectasy.

## DISEASES OF THE LIVER

Diseases of the liver are generally accompanied by gastric symptoms; with cirrhosis and the resulting circulatory disturbances of the viscera the gastric secretion (free hydrochloric acid) is more frequently diminished. The findings in other liver disturbances are not constant. Hematemesis may occur.

## ANEURYSM

In a case of aneurysm of the celiac axis, referred to under Cancer, free hydrochloric acid was absent, lacid acid present, and the stomach dilated. Circulatory disturbances were responsible for the gastric findings and pressure for the dilatation of the stomach.

## DISEASES OF THE KIDNEYS

Gastric disturbances are frequent in nephritis, and nausea and vomiting may be the first symptoms. In fact, Osler has reported death with these symptoms, and nephritis may be unsuspected until autopsy. The excretion of urea through the gastric mucous membrane or cerebral irritation from the poison are responsible for the vomiting. Variable conditions of the gastric secretion have been reported by various observers.

Biernaki[1] has studied 25 cases of nephritis, both acute and interstitial, and found in general the gastric secretion was diminished, and also that the quantity of free hydrochloric acid was reduced in proportion to the extent of the edema, the excretion of albumin, and the reduction in the quantity of urine excreted. Pepsin was reduced and the motor function was increased. Free hydrochloric acid was present in the mild cases in large or small quantities.

Einhorn has observed achylia gastrica in a case of renal calculus, which disappeared after operation, and A. A. Jones[2] has found achylia gastrica among patients with kidney disturbances.

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[1] Berlin. klin. Wochens., 1891, Nos. 25 and 26.

[2] N. Y. Med. Jour., Jan. 19, 1895.

## DIABETES

The digestion is sometimes good in diabetic patients, so that frequently the functions of the stomach are never examined.

Variable results, however, have been secured. Atrophy of the gastric mucosa has been found in a few cases. The motor power was good. The gastric motility is quite frequently markedly increased, so that it may be necessary to aspirate a test-breakfast within less than half an hour after its ingestion.

Rosenstein[1] reports normal secretion in 4 cases and abnormal in 6, while Gans[2] found 6 normal and 4 negative. Gilbride[3] finds that the secretion *of pepsin is frequently reduced* and sometimes absent; also that a high percentage of hydrochloric acid does not *indicate a good pepsin digestion.* Hydrochloric acid was absent in 3 of the 7 cases reported. The findings are not constant, and both normal and abnormal conditions of the secretion have been observed. Hydrochloric acid may be absent for a long time and then reappear. As most diabetics have arteriosclerosis, it is probable that the latter accounts for some of the changes in gastric functions in certain cases.

## ARTHRITIS DEFORMANS

In 1 case I found hyperchlorhydria marked; and Einhorn reports 1 case of achylia. The relation of intestinal putrefraction to arthritis deformans is described later under that topic.

## GOUT

In 2 cases Einhorn reports achylia, and in several mild cases hyperchlorhydria.

*Grip.*—Gastric disturbances are reported by Kaufmann in grip.

## MALARIA

Gastralgia may occur as a substitute for the malarial paroxysms and has already been described, or vomiting may be present, associated with malarial symptoms. There are no characteristic features of the gastric secretion, but in the latter cases free hydrochloric acid may be diminished.

*Arteriosclerosis.*—General arteriosclerosis may affect the gastric vessels and produce disturbances. Harlow Brooks refers to arteriosclerosis occurring chiefly in the abdominal vessels. The possibility of this latter condition should be considered (see Visceral Arteriosclerosis).

## DISEASES OF THE SKIN

**Eczema.**—Various systemic conditions probably have a bearing, and in some few cases correction of the digestive disturbances seem to have an influence in improving the condition. In one case I found hyperchlorhydria, and in another deficient hydrochloric acid.

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[1] Berlin. klin. Wochens., 1890, No. 15.

[2] IX Congress f. innere Medicin, 1890, Wiesbaden.

[3] Jour. Amer. Med. Assoc., Feb. 18, 1911.

Hyde[1] believes that gout, dyspepsia, constipation, and scrofula have a decided influence.

**Acne Simplex and Acne Rosacea.**—These are associated at times with gastric disturbances. Einhorn reports 2 cases of acne rosacea in whom chronic continuous gastrosuccorrhea was found. The correction of the latter benefited the skin affection.

**Psoriasis.**—The treatment of gastric disorders in this affection does not seem to benefit the lesion, according to Einhorn.

**Urticaria and Erythema.**—Some persons have an idiosyncrasy to lobsters, crabs, strawberries, etc., and develop therefrom poisonous substances which produce these eruptions, associated at times with acute gastric symptoms. These conditions seem to be due to auto-intoxication. Combe ("Intestinal Auto-intoxication") believes that acne, the seborrheic eczemas, urticaria, pruritus, strophulus infantum, and furunculosis to be chiefly due to intestinal auto-intoxication. He advises fresh brewer's yeast, 1 dram (4.0) t. i. d. before meals, for these conditions. Duncan Bulkley believes that cutaneous lesions are in some cases produced through cutaneous elimination of toxic substances. The author holds that the entire gastro-intestinal tract should receive attention. Thorough catharsis should be carried out. These foods should thereafter be avoided.

**Pemphigus of the Mouth.**—Einhorn has noted 3 cases in which there was hyperchlorhydria or neurasthenia gastrica, and in 2 cases improvement resulted from treating the gastric symptoms.

In general, we may say that considerable investigation is still necessary to definitely determine the relations of gastric disturbances to skin diseases.

## TUBERCULOSIS OF THE STOMACH

**History.**—Tuberculosis of the stomach is a comparatively rare condition. Arloing in 1903 collected 147 cases and Ricard and Chevrier in 1905 discussed 107 cases. Winternitz in 1908 refers to 7 cases of primary and 47 cases of secondary tuberculosis of the stomach reported up to 1901, and 9 or more secondary cases since that date. From these diverse statements, it seems difficult to decide on the authenticity of some of the reported cases. A. G. Ellis, in the New York Medical Journal, March 12, 1910, reports 2 cases. In the first patient, the condition was secondary to an extensive pulmonary tuberculosis. There were numerous gastric ulcers and a number of tubercles in the gastric mucosa. In the second patient, there were several ulcers, a number of tubercles, and a cyst containing tubercle bacilli. There was a chronic tuberculous peritonitis with adhesions. These cases were found at autopsy, as are most cases of tuberculous gastric ulcer. Tuberculous ulcers of the pylorus, with narrowing of the orifice, have been reported, and Ricard and Chevrier describe a sclerosing type of pyloric tuberculosis.

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[1] Twentieth Century Practice, vol. v.

**Anatomic Types of Gastric Tuberculosis.**—There may be a single ulcer or multiple ulcers. Diffuse miliary tuberculosis of the organ may be present and more rarely solitary tubercles. Tumor-like masses, usually at the pylorus, have been described. They may simulate carcinoma. Tuberculous cicatricial pyloric stenosis may occur.

*Diagnosis.*—The determination of pulmonary tuberculosis or a tubercular focus elsewhere is of value, as gastric tuberculosis is most frequently a secondary condition. Syphilis must be excluded by the Wassermann and Noguchi tests. A persistent afternoon elevation of temperature is suggestive. The *tuberculin test* should be made for diagnostic purposes.

*Symptoms.*—There are no specific symptoms of gastric tuberculosis. The tubercular ulcer is chronic, is not as apt to bleed as the simple ulcer, and the pain is liable to be severe in character.

*Treatment.*—Pulmonary tuberculosis or any other tubercular primary lesion should receive appropriate treatment. Carbonate of creosote or guaiacol carbonate, 5 grains (0.3) three or four times a day, are indicated for tubercular gastric ulcer; thiocol, 10 grains (0.6) t. i. d. is excellent. Also the bismuth preparations and proper feeding. Iron, arsenic, and the fats should be employed. Tuberculin may be employed cautiously by injection.

*Surgery.*—If a localized tubercular process, ulcer, or tubercular mass can be diagnosed, excision is indicated.

## SYPHILIS OF THE STOMACH

Gastric symptoms quite frequently occur in the secondary and tertiary stages of syphilis. In the secondary stage they may often be attributed to fever (the constitutional condition); while in the tertiary stage there are anatomic changes in the stomach itself.

Fenwick[1] believes that syphilis may affect the stomach in three ways: By the formation of gummata, by producing endarteritis, and by a chronic inflammation of the mucous membrane. The symptoms arising subside under antisyphilitic treatment.

Flexner[2] holds that syphilitic gastric ulcer is not rare, while Dieulafoy[3] notes various lesions, such as hemorrhagic erosions, ecchymoses, gummata, infiltration of the submucosa and circumscribed gummatous ulceration, and cicatrices of the latter. Pain, vomiting, hematemesis, and melena occur.

Riegel[4] reports 12 cases in which they complained of gas, nausea, distress after eating, and gastralgia, which responded promptly to antisyphilitic remedies.

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[1] London Lancet, Sept. 20, 1901.
[2] Amer. Jour. Med. Sci., Oct., 1898.
[3] Gaz. Heb. de Med., 1902.
[4] Diseases of the Stomach.

*Death* has resulted from perforation of *a broken-down gumma.*

All doubtful cases should be examined for signs of previous syphilitic infection,[1] for active syphilitic manifestion; also as to the history, and should be tested by specific treatment. Of course, many patients may have digestive disturbances without any connection with the old luetic condition.

Hemmeter[2] has described syphilis of the stomach.

Einhorn[3] gives the following classification and describes cases:

(1) Gastric ulcer of syphilitic origin.
(2) Syphilitic tumor of the stomach.
(3) Syphilitic stenosis of the pylorus.

To this I shall add a *fourth type:*

(4) Syphilitic cirrhosis of the stomach.

### (1) GASTRIC ULCER (SYPHILITIC)

A number of cases have been reported in which the usual treatment for ulcer failed, and which made complete recovery under specific treatment.

### (2) SYPHILITIC TUMOR OF THE STOMACH

This condition is excessivley rare. Einhorn has reported 2 cases, and refers to the fact that they may run their course like carcinoma. I referred to a case in this volume under Differential Diagnosis in Carcinoma of the Stomach. In this patient the gastric analysis showed absence of hydrochloric acid and lactic acid abundant. The patient had lost 77 pounds in eight months and was vomiting continuously. The stomach was dilated to below the umbilicus, and though he had been on specific treatment for a time before I saw him, the pyloric obstruction was so marked that drainage of the stomach was necessary to preserve life. Palpation gave a sense of resistance at pylorus.

A rapid laparotomy at the Red Cross Hospital disclosed a gummatous tumor at the posterior wall of the pylorus, nearly blocking it. Gastro-enterostomy was performed and specific treatment pushed. There was no more vomiting and the case has steadily improved.

### (3) SYPHILITIC PYLORIC STENOSIS

Einhorn reports a case of pyloric stenosis cured by antisyphilitic treatment. In most of these cases, however, gastro-enterostomy is required in addition to the antiluetic treatment.

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[1] The test for Wassermann's reaction is important, or Noguchi's test, both as confirmatory and for the diagnosis of doubtful cases.

[2] Diseases of the Stomach, p. 556, 1897.

[3] Ibid., p. 534, 1906.

### (4) SYPHILITIC CIRRHOSIS OF THE STOMACH

This case has also been referred to under Cancer. The stomach was small, hard, and contracted, and on palpation felt like a cirrhotic carcinoma, involving the entire stomach. The patient was an elderly man, had lost considerable weight, and was suffering from gastric symptoms.

Examination demonstrated cirrhosis of the liver and evidences of old syphilis. Deficient HCl, or achylia, occurs in this type.

These facts show that the syphilitic history, or evidences of the same, should be investigated carefully in gastric affections.

**Treatment** should be for syphilis. The author believes that mercury should be combined with the iodid treatment in these cases. For example, mercury salicylate in albolene (10 per cent. strength). Inject of this by hypodermic 10 minims every third or fourth day. In addition, preferably, potassium iodid, 30 grains to 1 dram (2.0–4.0), should be given daily in divided doses at meals. Twice the quantity may be administered by a gradual increase. It may be necessary to substitute sodium iodid. The injection of "606" may be employed. Appropriate remedies may be given in addition for special symptoms, or secretory or motor disturbances.

# PART III

# DISEASES OF THE INTESTINES

## CHAPTER XX

## METHODS OF EXAMINATION OF THE INTESTINES; EXAMINATION OF THE FECES; MECHANICAL PROCEDURES

**Special Interrogation.**—We presuppose that the method of interrogation of the patient, as described in Part I, has been carried out. Disease of the stomach may *produce secondary intestinal symptoms*, and hence the condition of this organ must be inquired into, and in most cases the functions examined.

Inquire, furthermore, whether abdominal pains are present, and also their position. In the right iliac fossa they suggest appendicitis or catarrh of the cecum; in the left iliac fossa, disturbances of the descending colon or sigmoid.

With rectal pain, inflammation in that region is probable, while pains near the navel usually originate in the small intestine. Pains of short duration and sharp in character are generally due to colic, and are followed and relieved by the passage of flatus or feces. They often shift from one region to another. Pains of long duration are usually from some *organic lesion*, such as ulcer or from some affection of the sensory nerves.

Abnormal sensations, such as feelings of heat or cold, may be experienced over different regions of the abdomen.

Tenesmus is present in dysentery and in many rectal affections. The time of the pain, whether immediately after meals or later, or during the night or in the early morning, is important.

The stool should be investigated—whether constipation, diarrhea, or alternating conditions exist, the number of movements, time of appearance, odor, color, and characteristics, as to whether mucus, blood, bile, pus, or undigested food are present.

Do climatic changes or mental excitement influence the bowel action, or are headache, dizziness, or exhaustion associated with the movements? Is there distention of the abdomen with gas, localized or general? When does it appear? Is rumbling (are borborygmi) present? Do belching of wind or passage of gas from the bowel occur, and does this give relief?

Total absence of flatus is important. Occurring with obstinate constipation, it would then suggest obstruction.

Continuous vomiting associated with intestinal symptoms suggests obstruction. With acute symptoms the temperature should be taken immediately.

## PHYSICAL EXAMINATION OF THE INTESTINES

The reader is referred to Chapter IV for general methods.

**Inspection.**—The retracted or trough-shaped abdomen occurs in stricture of the esophagus or cardia, basilar meningitis, lead-poisoning, and with long-continued inanition. The peculiar contour of enteroptosis has been described—the concave epigastrium, sulcus between the recti above the umbilicus, and the pot-belly below.

On distention of the normal colon with gas ($CO_2$), the ascending and descending portions are seen as elongated swellings in the lateral regions, and the transverse colon at or just above the umbilicus.

*Protrusion* of the abdomen may be over a definite area or over the entire surface. It may assume the shape of a rounded hemisphere, or oval, slightly flattened, especially in atonic conditions of the intestines and in hysteria. Marked uniform distention with tense abdominal walls, *absence* of respiratory abdominal movements, and increased thoracic respiration are present in peritonitis. There may be a general bloating with atony, but there is not the marked tension of the abdominal walls, and the other symptoms are absent.

With ascites the abdomen is evenly protuberant above, with the center somewhat flattened, while the lateral and dependent parts bulge *somewhat* in the recumbent position; change of posture alters the shape of the abdomen. This applies to the milder types.

With marked distention, as with meteorism, the enlargement is uniform. There is no bulging in any special location, except that the anterior portion is more prominent and change of position has no effect. Palpation aids under these conditions.

There may be protrusion of the abdomen in cases of neoplasm, in fecal accumulation, and occasionally in abscess, as of the appendix, from diverticulitis or other intra-abdominal suppuration.

Hernial protrusions at the umbilicus or in the inguinal regions may be observed.

In patients with thin abdominal walls, small sausage-shaped protrusions are occasionally visible, which change their shape and position. This is due to peristalsis of the small intestine, occurs with no pain, and denotes no morbid condition.

Similar waves may appear periodically and annoy the patient when caused by nervous influences.

There are sometimes violent contractions (peristaltic unrest) of the small intestine visible, caused by stenosis or obstruction. If it is near the ileocecal valve, the swollen and moving coils of intestine lie one above the other in the central part of the abdomen (ladder pattern). Intense pain accompanies these movements.

Marked distention may be visible in the course of the colon (in the circumference of the abdomen), and if associated with visible peristaltic contractions of the large intestine, passing along it from right to left, it is diagnostic of partial or total obstruction of the large bowel.

In some cases a recurring protuberance is noted, disappearing with a loud sound. This is probably near the point of stenosis.

## INSPECTION OF THE RECTUM—PROCTOSCOPY AND SIGMOIDOSCOPY

The anus can be inspected by having the patient lie on his side with thighs and knees flexed, and his back toward the examiner. The buttocks should be held apart with the hands. Hemorrhoids, fissures, and fistulæ may be discovered.

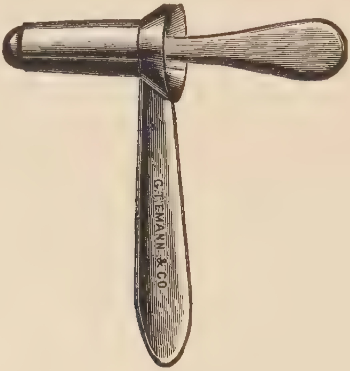

Fig. 195.—Kelly's short rectal speculum.

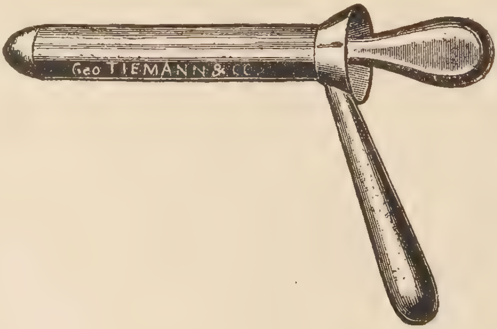

Fig. 196.—Kelly's standard rectal speculum.

For inspection of the rectum the introduction of a speculum (proctoscope) is necessary.

**Proctoscopy.**—Various instruments have been devised, notably those of Howard Kelly, Roberts, Sims, Kelsey, Gant, and J. P. Tuttle (Figs. 195–203).

Roberts'[1] glass speculum may be of value for the examination of a patient with an irritable anus. The instrument does not distort the parts and can be readily introduced. There is a lateral opening for the purpose of making an application or employing a probe. There is a mirror so arranged that it can be pushed in, withdrawn, or rotated, and so give a view of the entire wall at a varying depth. It is necessary to use reflected light from a head-mirror or an electric head-light (Fig. 197).

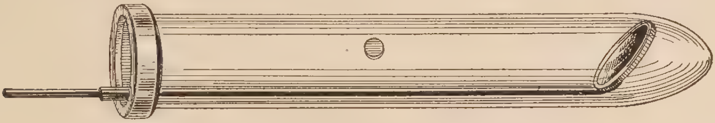

Fig. 197.—Roberts' glass anal speculum.

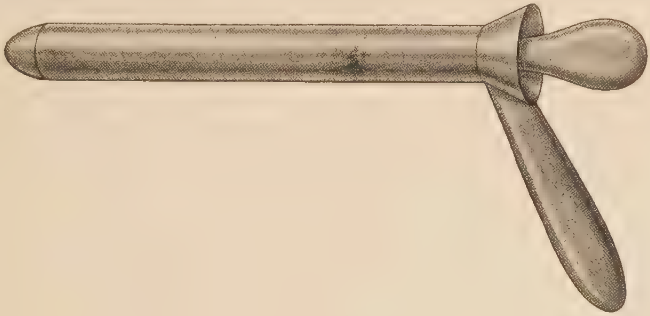

Fig. 198.—Kelly's proctoscope ($\frac{1}{3}$ actual size).

The bowels should preferably be thoroughly evacuated by enema before the examination. If the region is sensitive, a few drops of a 2 to 5 per cent. cocain solution can be injected inside and along the sphincters with a narrow-pointed rubber syringe. A suppository containing opium, 1 grain (0.065), with extract of belladonna, $\frac{1}{3}$ grain (0.022), or cocain, $\frac{1}{8}$ grain (0.008), can be substituted.

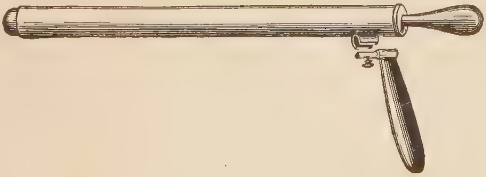

Fig. 199.—Kelly's sigmoid speculum.

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[1] Jour. Amer. Med. Assoc., Jan. 8, 1910.

Tuttle's pneumatic proctoscope is a valuable instrument. There is an electric lamp at the end of the inspection tube and an arrangement for inflation of the rectum, so that it can be distended with air at the time of examination.

With other specula, a head-mirror with electric-light attachment is most convenient, though an ordinary light can be arranged. The patient lies on the side, with thighs and knees flexed, with back toward examiner, or the legs can be elevated on a crutch, or in some cases the knee-chest position can be assumed. The external sphincter as well as the speculum should be lubricated with sweet oil or vaselin, to render introduction more easy.

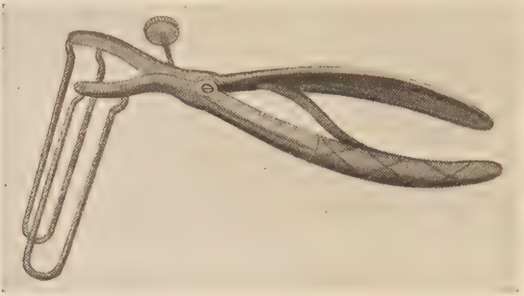

Fig. 200.—Sims' rectal speculum.

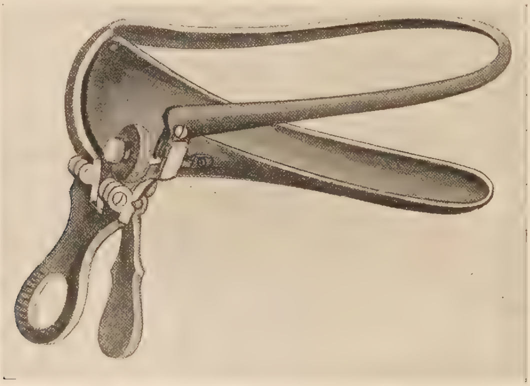

Fig. 201.—Kelsey's speculum.

**Sigmoidoscopy.**—A long speculum (sigmoidoscope) may be required for examination of the sigmoid (Figs. 204 and 205). Among the best sigmoidoscopes are those of Kelly, Tuttle, and H. Strauss; Beer[1] reports a modification of the latter. My own preference is the pneumatic sigmoidoscope of Tuttle or Strauss. Strauss' instrument possesses the advantage that if the lamp becomes dirty, it can be removed and cleaned without taking out the entire instrument, and also has a pneumatic inflator, as has Tuttle's sigmoidoscope. Tuttle introduces the instrument in the Sims posture, while Strauss advocates the knee-chest position. In Fig. 206,[1] arrow No. 1 shows the direction of the apparatus at the beginning of the entry; arrow No. 2, the position at the view of the ampulla recti, and arrow No. 3, the direction at the time of entering the flexure of the colon (sigmoid). The presence of blood, or blood and pus in the stools, or progressive constipation are indications for examination of the rectum and sigmoid. One should, of course, exclude ulcerations of any portion of the small intestine. When rectal examination (proctoscopy) fails to afford the desired information, sigmoidoscopy is indicated. One should, of course, acquire all possible information by abdominal palpation and other methods.

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[1] N. Y. Med. Rec., Feb. 11, 1911.

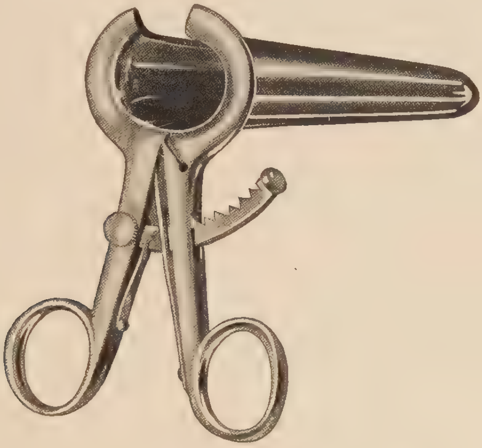

Fig. 202.—Gant's examining speculum.

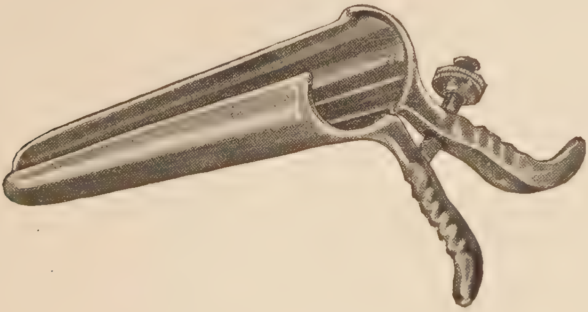

Fig. 203.—Gant's hinged speculum.

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[1] S. Kelen, Pester Medizinische-chirurgische Presse, April 17, 1904.

**Palpation.**—The technic of simple and reinforced palpation has been described in Chapter IV.

The cecum, parts of the ascending and descending colon, the transverse colon, and the sigmoid flexure are often accessible to palpation, but not as readily so in obese or in muscular subjects. Fecal accumulation, tumors, thickening of the gut, or purulent collections connected with the intestine can often thus be recognized.

An uneven protuberant surface is characteristic of malignant growth, while an even surface is more often found in benignant neoplasm or intussusception. Volvulus occurs usually in the sigmoid. A fecal accumulation will, as a rule, " pit " on pressure (give a doughy feel). Hard scybalæ occasionally feel like marbles under the fingers, but can be moved or slightly indented. Sometimes when raising the fingers from palpating, there may be a crepitating or sticky sensation, or the intestinal wall can be felt to slip off from the fecal mass. This symptom was first described by Gersuny.[1]

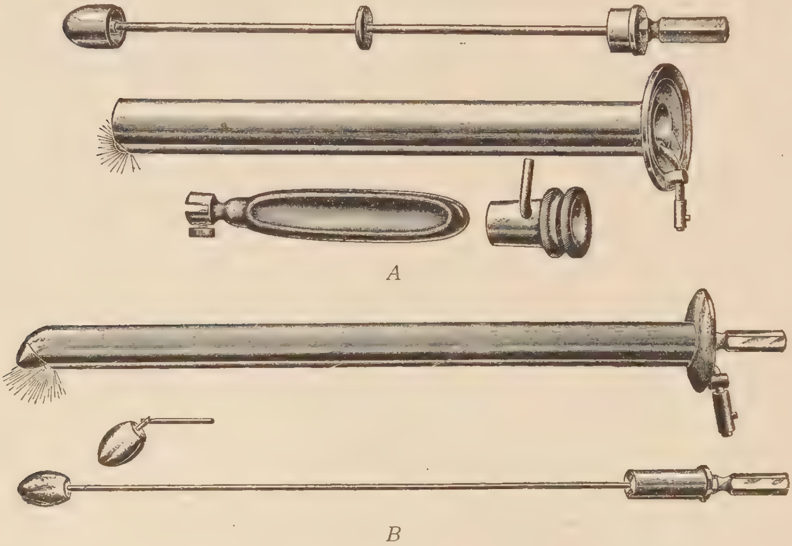

Fig. 204.—*A*, Tuttle's pneumatic proctosigmoidoscope. Two-lengths—rectal 4 inches, sigmoid 10 inches—with window attachment to make instrument air-tight for bowel inflation; *B*, Tuttle's sigmoidoscope with Mercier curve.

Gurgling occurs in typhoid on palpation in the right iliac fossa, but is not diagnostic.

Tenderness or pain on pressure can be readily determined by palpation, and are suggestive of inflammatory processes or ulceration. There may be the general tenderness of acute intestinal inflammation or the acute pain and tenderness of peritonitis, either localized or general.

[1] Wiener klin. Wochens., 1891, No. 4.

Circumscribed pain on pressure may be present at McBurney's point (1½ to 2 inches to the inner side of the anterosuperior spine of the right ileum) on a line drawn from this process to the umbilicus.[1]

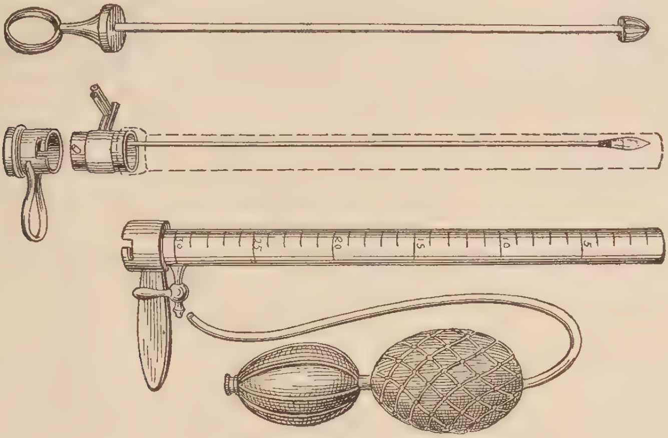

Fig. 205.—Strauss' sigmoidoscope.

With ulceration of the bowels there may be circumscribed areas very sensitive to pressure; with hysteric manifestations there are often sensitive spots complained of in the abdomen, as, for example, in mucous colic. By palpating simultaneously two distinct points, the supposed painful area and another region, with the different hands, and at the same time distracting the patient's attention by conversation, one often finds an absence of true tenderness at the supposed seat of pain.

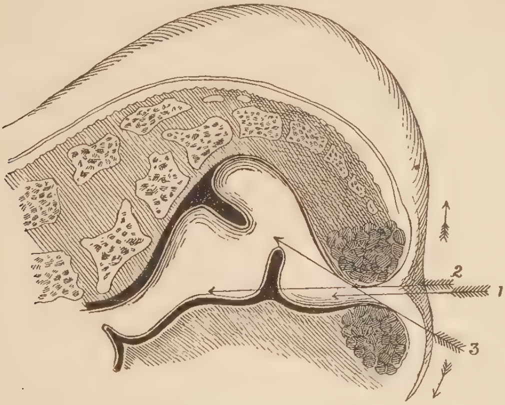

Fig. 206.—Sigmoidoscopy (knee-chest posture, Strauss): Arrow 1 shows the direction of the apparatus at the beginning of entry; arrow 2, the position at the view of the ampulla recti; arrow 3 shows the manner of entering the flexure.

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[1] Morris' point and the lumbar ganglia are referred to under Appendicitis.

Muscular rigidity shows peritonitic involvement. It may be localized, as of the right rectus in the region of the appendix or gall-bladder, or over the left rectus, as in diverticulitis or phlegmonous gastritis, or in abscess of the left lobe of the liver. General rigidity shows general peritonitis.

**Splashing Sound (Clapotage, Succussion).**—If the intestines contain liquid material and gas, tapping over them with the fingers will at times produce the splashing sound. The method of differentiation between stomach and intestinal splash has been described under Splash of the Stomach in Chapter V.

In the small intestine clapotage can usually only be obtained in the dilated portion of the gut above a stricture. It is not uncommon in the large intestine, and can be most often determined in the sigmoid flexure, caput coli, and the transverse colon. In case of atony of the bowel it is quite frequently present, also in the relaxed abdomen of enteroptosis, and often in patients with hysteric manifestations.

Boas[1] first suggested injecting into the bowel 1 pint (500 c.c.) or more of water and then examining for the splash along the colon. It should be given with *hips elevated.* The splash will first be secured in the sigmoid, and by turning the patient on the right side it can at times be produced in the transverse colon and in the cecal region.

It is possible to administer an enema of moderate size and cause it to gravitate to the caput coli by the method of rotation described under Enteroclysis. By the splash one can determine whether an injection given for dysentery has passed through the entire colon to the cecum.

In atony of the bowel, Boas produced the splash, even after the injection of only 6 to 10 ounces (200–300 c.c.) of water. The position of the colon can be determined by the splashing sound when it is present; it can be produced artificially by the injection of water into the bowel, and be thus employed for locating position of the intestines. A little Vichy, 4 ounces (125 c.c.), can be added to the injection to increase the amount of gas.

**Palpation of the Rectum.**—This procedure, the *author believes, should be carried out, before* inspection with the *proctoscope.* The rectum is preferably palpated with the index-finger. Soap should be placed under the edge of the nail to prevent fecal material lodging therein, and the finger lubricated with vaselin or olive oil. It is well to grease the external sphincter, as it renders entrance of the finger easier. It is more cleanly to encase the finger with a thin rubber cot, or to employ a rubber glove, well lubricated.

The patient lies on the side, with knees flexed and the back to the examiner, or he may be in the knee-chest position, or stand with the waist flexed—leaning forward over a chair and bearing down as if to defecate. Hemorrhoids, polypi, low-seated stricture, tender points suggestive of ulcer or fissure, malignant growths, rectal prolapse, abscess, fecal obstruction, foreign bodies, and intussusception are often within reach of the examining finger. The prostate or uterus should be palpated during the examination. If the difficulty is located beyond reach of the palpating finger, *inspection* with the proctoscope or sigmoidoscope will give the required information. Simon's method of dilating the sphincter under anesthesia and passing the hand and arm into the bowel, for the purpose of palpation, *is a most dangerous procedure.*

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[1] Diagnostik und Therapie der Magenkrankheiten, 1897.

*Vaginal Examination.*—The author recommends *that in every case* the *female pelvic organs* should *be also examined.* Irritation or inflammation of the pelvic organs may have a reflex effect in producing irritation of the rectum and vice versâ. Traumatism or inflammation of the one are liable to extend to the other directly or through their lymphatic connections. An overweighted uterus may sag down on the rectum and produce hemorrhoids. Many other examples can be given. Pelvic and sacral pains, reflex pains, and irregularity of functions, in fact, very similar symptoms, may occur from either rectal or pelvic disease. The primary cause should be carefully investigated, and hence the additional value of a vaginal examination.

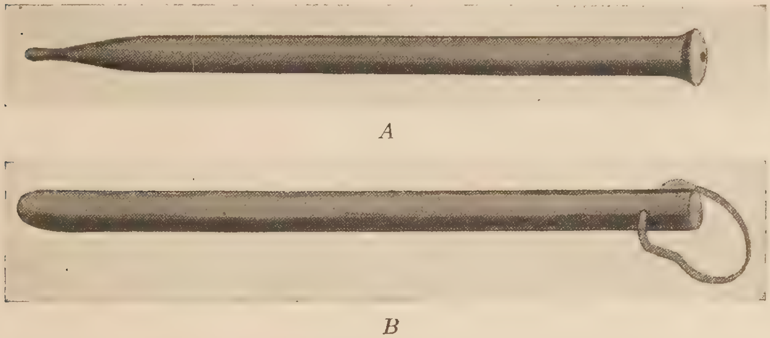

Fig. 207.—*A*, Soft rectal bougie; *B*, cylindric bougie.

*Palpation of the Rectum by Sounds.*—This is indicated when there is suspicion of stricture in the bowel not accessible to the fingers. Soft rectal tubes of various caliber may be employed. When the obstruction stops the passage of the tube, a mark is made at the external sphincter, so that the distance of the stricture up the bowel can be estimated. Smaller tubes are then employed until one can pass the obstruction. Its caliber is thus estimated. The ordinary soft flexible rectal or colon-tube is the safest for diagnostic purposes in the hands of the general practitioner.

In Fig. 207, *A* and *B*, are shown an olive-pointed flexible and a cylindric bougie. The latter is somewhat stiff and can be softened in hot or boiling water before use. This last is also employed for dilatation of the stricture.

Kuhn's metal spiral tube is of no advantage. Care should be exercised if stiff tubes are employed.

**Percussion** should be gentle. Over empty intestinal coils or those containing gas or air, a tympanitic sound results, which is louder over the large than over the small bowel. As there may be considerable distention of the small intestine, it is sometimes difficult to delimit the large intestine by simple percussion. If the colon is emptied by enema, and then distended artificially with air or carbonic acid gas, the procedure is much easier.

Intestinal coils which are filled with liquid or solid material give dulness on percussion. With meteorism there is tympanites of a deeper pitch than normal, and sometimes there is a metallic sound with auscultatory percussion. The meteorism may be localized or general. If local, in connection with visible peristalsis, intestinal stenosis is at once suggested. With local meteorism there will be dull areas elsewhere; with general meteorism the entire abdomen is symmetrically distended, the anterior portion being most protruded, and there is the diffused tympanitic note of the peculiar type noted, and dulness over the region of the liver and spleen may disappear. With ascites, percussion shows dulness in the lower lateral regions of the abdomen and tympanites in the middle. The sounds change on altering the position of the patient (turning him on his side).

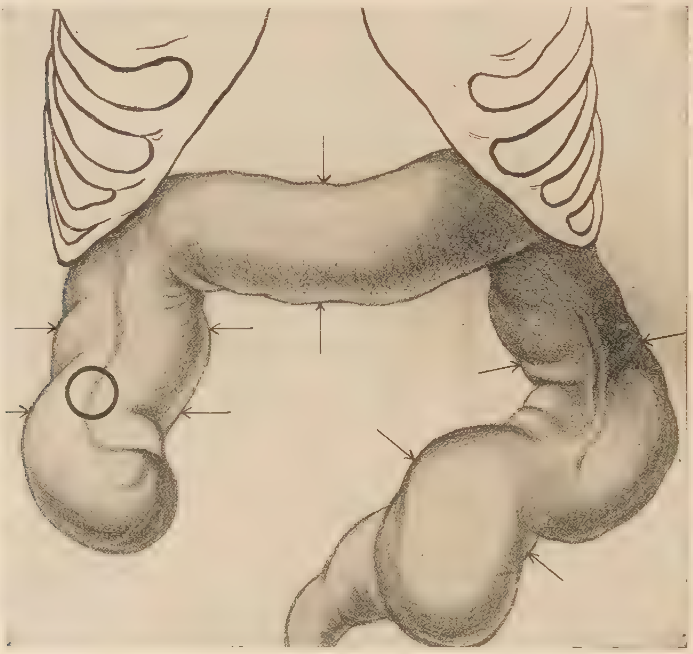

Fig. 208.—Auscultatory percussion of the colon.

The intestines ride up on the fluid, and the upper flank, previously dull when in the dorsal position, is now tympanitic.

Fecal accumulation, tumors, and abscesses give dulness on percussion.

*Auscultatory Percussion.*—This is the best method of determining the position of the colon. If the small intestine is excessively distended, it is difficult to differentiate the percussion sounds. It may be necessary to empty the colon by enema or irrigation and then inflate with air or $CO_2$. Place the stethoscope at the circle (Fig. 208) over the cecum; begin percussion midway between the umbilicus and symphysis, and percuss to the right, to the left, and upward, in the direction of the arrows, until in each direction the greater intensely altered quality and heightened pitch show that the inner border of the colon has been reached. These points can be marked on the abdomen with a pencil. Then percuss in the epigastric region (midway between the ensiform and umbilicus) downward, and from the lateral lumbar regions inward. The changes in pitch, quality, etc., should be marked, and thus the outer limits of the transverse ascending and descending portions of the colon are determined.

The scratch method of auscultatory percussion may be carried out in the same lines.

Stengel claims that by auscultatory percussion it is possible to determine that a tumor found to lie in the course of the intestine originates in the wall of the colon. In Fig. 209, C represents the tumor; the circle O, the stethoscope placed over the colon near the tumor; B, percussion note over small intestine; A, percussion over normal large intestine near the stethoscope.

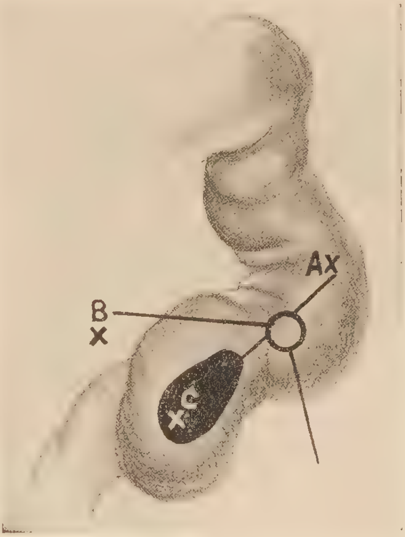

Fig. 209.—Differential percussion.

First percuss directly over the tumor, then toward it from every direction. The note over tumor, C, if it is connected with the colon, resembles the percussion note at A (colon) more closely than does percussion note at B (small intestine) resemble note at A (colon).

**Auscultation.**—This is not of great significance in intestinal diseases. Palpation may elicit a gurgling noise in the right iliac fossa, formerly thought to be pathognomonic of typhoid, but it is found in many other conditions. Gurgling sounds (borborygmi) are at times heard. They may be due to fermentative processes or occur in neuroses, and are not specially significant.

The entire absence of intestinal sounds may be significant of intestinal paresis. If the latter is due to peritonitis, cardiac and respiratory sounds may be audible over the entire abdomen. Friction sounds from perisplenitis or perihepatitis may rarely be auscultated. With chronic stenosis very loud noises are at times heard, caused by the sudden passage of fluid and gas through the stricture under pressure.

Splashing sounds are at times distinguishable in the enlarged bowel above the stricture.

## TRANSILLUMINATION OF THE INTESTINES

This method was first suggested by Einhorn,[1] and further practised by Heryng and Reichmann.[2]

Einhorn's technic is as follows: A high enema or irrigation of the bowel is first given. A quart of water is later injected per rectum, and the illuminator, very similar to the gastrodiaphane, is inserted and gradually pushed up the intestine. The examination must be made in a dark room. Experiments were carried out by the author with specially devised illuminators at the Manhattan State Hospital.

As the average adult rectum is 8 inches long, the sigmoid flexure 17½ inches, and allowance must be made for the sphincters, it requires an instrument *at least 30 inches in length* to pass through the sigmoid into the descending colon. The 30-inch instruments, with which I experimented, on almost every occasion caught and coiled back, and rarely did I succeed in securing transillumination of the lowest part of the sigmoid, and even then the light was so faint that it was entirely unsatisfactory.

Inflation of the bowel with water, with fluorescein solution, and with air were all tried before passage of the light, as were various positions of the patient.

The experiments demonstrated practically the impossibility of passing a flexible instrument or tube through the sigmoid. The sigmoid is quite movable, and Howard Kelly has shown that the colon-tube readily pushes it up. My experiments demonstrated visually the impossibility of passing the long colon-tube through the sigmoid flexure, and showed that transillumination of the sigmoid has not been sufficiently certain to prove of practical value.

**Röntgen Rays** (*x*-**Rays**).—For examination of the intestines the the *x*-rays are of value for the following conditions:

The determination of the presence of a foreign body in the intestinal tract, accurately locating its position, and hence the site for operation.

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[1] N. Y. Med. Montasschr., Nov., 1889.

[2] Therapuetische Monatshefte, 1892.

Einhorn[1] has recommended the internal administration of bismuth subnitrate in watery solution to locate the constriction in acute intestinal obstruction with the aid of the *x*-rays. The delay necessitated would be dangerous.

For locating the seat of chronic intestinal occlusion the method may be of service.

An ounce (30.0) of bismuth subnitrate or bismuth subcarbonate, preferably, can be administered in 12 ounces (375 c.c.) of milk or water, and about twenty-four hours later examination should be made with the *x*-rays. With the fluoroscope or by a photograph one would see a distended area of intestine filled with bismuth, a region below with apparently no bismuth or a trace (the point of stenosis), and below this a small amount of bismuth that had passed through the stricture. If the stricture is apparently in the large intestine, a check test can be made as suggested by Einhorn.[2]

A few days later, when the bowel is free from bismuth, an injection per rectum is given of 1 pint (500 c.c.) of water containing 30 grams (about 1 ounce) of bismuth subnitrate. If the stenosis is in the large intestine it will be located by the Röntgen picture, there being the area apparently free from bismuth (the stricture) and the collections above and below the stricture.

*Location of the Colon by the x-Rays.*—Two quarts (liters) of water, in which 60 grams (about 2 ounces) of subnitrate of bismuth or bismuth subcarbonate are suspended by means of a little starch solution, are injected per rectum, with the hips elevated or in the knee-chest posture. The position of the colon can be immediately determined by the Röntgen ray.

One could administer the bismuth by mouth and examine at the end of twenty-four hours, but the enema method is preferable. Misplacements of the colon, enteroptosis, and angulations of the sigmoid can thus be determined.

It has been claimed that a soft tube, in which lies a flexible wire, can be introduced per rectum, and the course of the colon determined by the *x*-rays, the wire showing a shadow. It is practically impossible to insert the tube beyond the sigmoid, so the method is not accurate. The *x*-ray pictures shown of the long colon-tube—*supposedly in the descending colon*—are usually in the ampulla of the rectum. H. W. Soper[3] has demonstrated, by means of the *x*-rays, that it is impossible to pass the colon-tube into the sigmoid, except in the case of Hirschsprung's disease (congenital idiopathic dilatation and hypertrophy of the colon).

As a *general method for locating the position of the colon* the Röntgen rays are expensive and often impractical, as they give no more information than by inflation. In *obscure cases*, or when *angulations, adhesions causing stricture*, etc., are suspected, they *are of value.*

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[1] N. Y. Med. Jour., May 18, 1907.

[2] Ibid.

[3] Jour. Amer. Med. Assoc., Aug. 7, 1909.

*Physiologic Investigations with Röntgen Rays.*—Cannon[1] has investigated intestinal movements after administration of bismuth by means of the *x*-rays. He experimented on cats, and claims there are periodic antiperistaltic movements in the cecum and ascending and transverse colon which aid in churning and mixing the contents, and so help absorption. He holds that some of the material may be forced back into the small intestine, and that this may occur with a high nutrient enema. The movements of the small intestine have also been studied by this method.

Some investigators disagree with Cannon. Grützner has shown that starch granules, lycopodium, powdered carbon, etc., in physiologic salt solution, injected into the bowel under favorable circumstances, will find their way upward into the stomach.

By means of the *x*-rays Hemmeter observed that the upward movement of these particles goes on simultaneously with the downward movement of the feces, *i. e.*, there is upward marginal current. He considers the epithelia and muscularis mucosa instrumental, and that it is not true antiperistalsis.

## INFLATION OF THE INTESTINES WITH CARBONIC ACID GAS OR AIR

Von Ziemssen[2] first employed inflation of the colon for diagnostic purposes by injecting in succession into the bowel solutions of tartaric acid and sodium bicarbonate, with the resulting development of carbonic acid gas. The bowel could then be recognized by the marked tympanitic sounds on percussion or, more rarely, by inspection. Preferably, the gut should be previously emptied by enema.

Inject into the bowel 1 dram (4.0) of tartaric acid, which has been dissolved in 6 to 8 ounces (200–250 c.c.) of water, and follow it by the injection of the same quantity of soda bicarbonate in same amount of water. Preferably the hips should be elevated.

Schnetter suggested attaching a flexible tube with rectal tip to the nozzle of an inverted soda-water siphon and driving out the $CO_2$ by pressing the valve.

The gas has also been injected by obtaining it from the liquefied gas in a sparklet and conducting the $CO_2$ from a bottle into the rectum.

One of the *simplest methods to inflate the bowel with carbonic acid gas* is by Rose's gas bottle. This consists of a moderate-sized bottle with perforated cork, through which passes a glass tube. To this is attached a soft-rubber tube and rectal tip (Fig. 210).

The bottle is half-filled with water and 1 dram (4.0) each of tartaric acid and soda bicarbonate added, and the cork tightly inserted. The accumulating gas is conducted off by the tube into the rectum. Inspection and percussion will determine the extent of distention.

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[1] Amer. Jour. of Physiol., vol. vi, p. 253.

[2] Deutsch. Archiv. f. klin. Medizin, 1883, Bd. 38, S. 325.

Runeberg[1] recommended inflation of the intestines by air by means of a colon-tube, to which a compressible air-bulb is attached. It is possible by this means to measure and regulate the quantity of air employed for inflation.

An ordinary Davidson's syringe can be used to pump in the air, and its capacity can be determined as follows:

Take a measuring glass of 1 pint (500 c.c.) to 1 quart (liter) or a glass vessel of unknown capacity and measure its capacity when filled to the brim. Invert the filled vessel in a pail of water, so that the entire column of water is sustained in the inverted vessel. Then slip the colon-tube under water, so that its tip enters the inverted jar. Observe how many compressions of the bulb are required to replace the water column with air, that is, to drive out all the water from the inverted vessel.

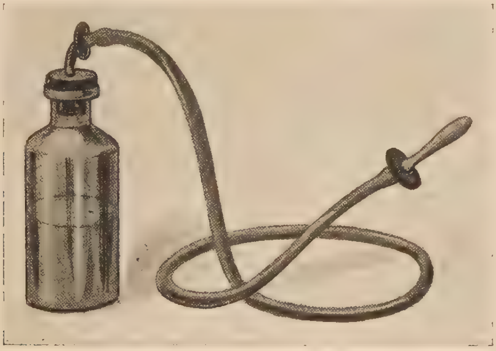

Fig. 210.—Rose's carbonic acid gas generator.

If, for example, the jar contained 1 pint (500 c.c.) and it required sixteen squeezes of the bulb to replace this by air, then each squeeze of the bulb replaces 1 ounce of water by the air equivalent.

**Uses of Inflation.**—It is of service to detect stenosis of the large intestine. Under normal conditions the injected air distends the colon evenly. If there is stenosis, the air will distend chiefly that part of the bowel below the stricture, while above it remains unchanged. This is true in marked strictures, but in those of mild type the air will pass through. Even in some such cases there will be less distention above than below the strictured point.

The *position* of the colon can be determined by air inflation. Normally it passes with a slight downward curve across the abdomen with lower edge about touching the upper margin of the umbilicus, or it may even lie just below the latter.

Enteroptosis is demonstrated by this method, and the transverse colon may descend to a hand's breadth above the symphysis. It may assume a V shape. Angulations of the sigmoid may at times be determined by this means.

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[1] Deutsch. Archiv. f. klin. Med., Bd. 34, S. 460.

For the diagnosis of the location of abdominal tumors inflation of the intestine is often of service. After inflation of the colon with air, tumors of the viscera become more distinct; while tumors of the kidney, retroperitoneal glands, spine, etc., tend to disappear.

Minkowski[1] holds that after filling the colon with air or water abdominal tumors are shifted in the direction of the organ to which they belong.

Sutton[2] suggests inflating the bowel with air impregnated with ether for the diagnosis of intestinal perforation. He employs a bottle provided with a perforated rubber cork to which are attached two rubber tubes with stop-cocks. To one of these tubes is attached a bicycle pump or a Davidson's syringe; to the other, an ordinary colon-tube by means of a short glass connecting tube.

Two drams of ether are placed in the bottle. The air pumped into the bowel passes through the bottle and thus takes up the vapor of ether. With the stop-cocks the pressure of ether and air can be regulated.

If perforation of the bowel is present, the ether escapes through the opening into the abdominal cavity and distends it equally in all directions. If there is no perforation, first the large intestine and later the small intestine become filled with air and ether, and, finally, ether vapor may be eructated and readily recognized. This method is useful in gunshot wounds of the abdomen.

**Inflation of the Colon with Water.**—This can be performed with a graduated irrigating jar and a rectal tube. With a fountain syringe of known capacity it is easy to estimate the quantity injected.

In *stricture of the colon*, especially in the lower portion, the quantity of water which can be injected is not great. Normally the colon will contain from 3 to 4 quarts, or occasionally 5, without dangerous distention. The injection should be given with hips elevated. *Many people are unable to hold any quantity of water in the bowels without pain, discomfort, and rapid evacuation of the fluid.*

Determination of the position of the colon by inflating it with water is, therefore, often difficult. The air inflation is preferable for the above reasons.

## LAVAGE OF THE BOWEL FOR DIAGNOSIS

Boas[3] first recommended this procedure to be carried out in a manner similar to lavage of the stomach. The bowels should be previously evacuated. The patient should lie on the side with the knees and thighs flexed. The colon-tube is attached by a short piece of metal or glass tubing to a long tube, provided with a funnel. The rectal tube is lubricated and inserted high up to its full length, and about 1 pint (500 c.c.) to 1 quart (liter) gradually poured in through the funnel held 1 or 2 feet above the patient, until some discomfort is expressed. The funnel is then lowered below the anus and the contents siphoned off.

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[1] Berlin. klin. Wochens., 1888, No. 31.

[2] Jour. Amer. Med. Assoc., Dec. 30, 1899.

[3] Deutsch. Aertze-Zeitung, 1895, Nos. 2 and 3.

They are then submitted to a thorough examination. Normally the contents are fairly clear or slightly stained with fecal matter, and contain a little mucus and a few epithelial cells.

With intestinal catarrh a large amount of mucus is present. Blood or pus may be found, showing hemorrhages or a suppurative process, such as an ulcer or abscess. Exfoliated portions of the mucous membrane may occasionally be found or, rarely, tumor fragments.

Microscopic examination of such material is of diagnostic importance. Intestinal worms are occasionally discovered. Lavage of the bowel is also employed for the removal of dysenteric discharges and mucus, for the purpose of examination for amebæ.

## EXAMINATION OF THE FECES

**The Stool.**—*General Considerations.*—The normal stool consists of changed and unchanged remnants of food, bacteria (estimated at about 126,000,000,000 daily), epithelial cells, salts, and traces of the digestive juices.

The normal daily quantity under a mixed diet averages 100 to even 200 grams (3½ to 7 ounces). It may be increased by a vegetable diet. There is usually one movement daily of dark brown color, though diet and medicine have an influence. Milk gives a light yellow; claret and huckleberries, a brownish black; salts of iron and magnesia, a blackish brown; bismuth, black. Blue is given by iodids (long continued); green, by calomel; yellow, by santonin, senna, and rhubarb; violet, by salol and betanaphtol. The feces are slightly soft and of sausage shape. Abnormally they may appear in small balls, cylinders or tape-like, or as hard scybalæ (dry in character), or they may be mushy or liquid. They may be very watery, as in choleraic conditions, or fluid and mixed with mucus.

*Odor.*—This is normally caused by skatol and slightly by indol. It is increased when the feces have been retained an abnormal time. After a short sojourn in the intestines, as with rice-water movements, there is often no odor. The character of the food may affect the odor. Very fetid movements occur with ulcerative processes or with malignant growths.

**Macroscopic Findings.**—*Remnants of Food in the Feces.*—Undigested remnants of food can often be seen in the stool. Normally they consist of only small particles of vegetable material, such as potato, asparagus, spinach, and peas; while *remnants of meat* cannot be seen. Providing abnormal quantities of food have not been ingested, it is often possible to draw definite conclusions as to the state of intestinal digestion, from the excess of one form of non-digested material over another. The presence of *large quantities of undigested starch indicates a catarrhal condition of the small intestine*, and, indeed, more *than traces* of this material should be *regarded with suspicion.* If particles of meat are visible, this indicates a lesion of the intestinal tract. *Connective-tissue fibers* appearing unaltered in the feces indicate deficient gastric digestion, according to Schmidt, and the presence of nuclei, under the microscope, disturbance of the trypsin function of the pancreas.

*Blood.*—Blood may be visible in the feces, either fresh (red) or dark in color and uncoagulated, which shows its origin from the lower part of the large bowel. It may appear changed, giving the feces the appearance of tar, then originating from the small intestine or even from the stomach. Blood shows the presence of an ulceration or of an ulcerating cavity communicating with the gut.

*Pus.*—Visible pus in the dejecta only occurs when pus exists in large quantities in the lower part of the large intestine. It shows ulceration or an abscess communicating with the gut. Pus in small quantity or from higher up the intestine can only be determined by the microscope.

Fragments of tumor (polypi or cancer) may rarely be found in the dejecta. Microscopic examination will give important information.

*Mucus.*—Mucin can always be detected in normal feces by chemic examination. The amount of mucus in the feces in health is so small and so intimately mixed as to be only recognized by chemic tests. Mucus in the stool, either macroscopic or microscopic, indicates some deviation from *the normal physiologic condition.* It does not *invariably show an anatomic lesion.* We may, for example, have a few flakes of mucus or an extremely thin layer adherent to scybalæ, due to irritation of the mucosa from a fecal accumulation or impaction, or mucus, which is contained normally in the higher portions of the small intestine, may occasionally appear in the stool as a result of increased peristalsis.

Under other circumstances the presence of mucus is pathologic. Macroscopically, mucus may exist as follows:

(1) An abundant coating in the form of a glassy layer may cover fecal masses. It may be gray or cloudy from epithelial or round cells. This usually indicates catarrh of the lower portion of the bowel.

(2) It may be intimately mixed with the feces in mushy movements, and may adhere to a glass rod if this is dipped in the stool.

(3) It may float on top of watery movements.

(4) It may be passed almost pure in large amount.

Material resembling *frogs' spawn* or sago grains may occur in the feces. They were formerly considered due to follicular ulceration, but are now believed to be of vegetable origin. Kitagawa holds that some of them are pure mucus, but that they are not pathologic.

Yellow or yellowish-brown granules occur in the stool, from the size of a pinhead to a poppy seed, resembling butter in consistency. Some of these have been considered bile-stained mucus, while other fragments are believed to be albuminous or vegetable material or yellow calcium salts.

Boas and Schmidt believe these yellow granules to be albuminous matters stained with bile-pigment.

The presence of any one of the previously described types of mucus indicates intestinal catarrh. Mucus without feces or surrounding the feces shows the colon is inflamed. When mucus is mixed with the feces the upper colon or small intestine is inflamed. Mucus in the food residue shows catarrh of the small intestine.

There are two exceptions to the rule that *visible mucus indicates catarrh:*

(1) In mucous colic (membranous enteritis) pure mucus due to hypersecretion is evacuated in the form of a cast membrane or in long tape-like formation.

(2) In intestinal dyspepsia with acid fermentation the patient has a jejunal diarrhea, with gelatinous, tenacious, semifluid stools. Mucus is present.

Epithelial or round cells, which are abundant in *catarrhal mucus*, are *absent* from the mucus in dyspepsia of the small intestine. The stools are also green, acid, and give a bile-pigment reaction.

*Intestinal parasites* may be visible in the feces.

**Chemic Examination of the Feces.**—*Reaction* is normally *neutral or slightly alkaline.* Marked acidity results from occlusion of the bile-duct. Rich vegetable diet causes slight acidity. The simplest method to test the reaction is by litmus-paper (red and blue).

Normal stools react slightly differently with different indicators. With phenolphthalein they react slightly acid, while to litmus they would be neutral. If the phenolphthalein test is employed, take feces 5.0 c.c., rub up in a mortar, and add 30.0 c.c. distilled water, after the modified Schmidt diet. Place 2 c.c. of this in a test-tube, add 2 drops of 1 per cent. alcoholic solution of phenolphthalein. With this quantity titration with decinormal sodium hydrate never exceeds 1.5 c.c. to secure end-reaction. Above this the stool should be considered acid, and if less then 1 c.c. it may be considered alkaline (Kaplan[1]).

For general use the litmus test is sufficient.

*Test for Mucin.*—Mucin is normally present in the feces. Mix feces with water and an equal quantity of milk of lime and let the mixture stand for several hours. Then filter, add acetic acid to filtrate, and mucin precipitates if present.

*To Examine Separate Particles of Suspected Mucus.*—Dissolve a flake of material in a weak solution of potassium or sodium hydroxid and add acetic acid. If the precipitate is undissolved after adding the acid in excess, mucin is present. Heat the precipitate to the boiling-point in a dilute mineral acid; if mucin is present the heated solution will reduce copper oxid. This last test excludes nucleo-albumin, which otherwise gives a similar reaction (Einhorn). Stain a flake of apparent mucus with a weak triacid solution (Ehrlich), mucus produces a green color; albumin, red. This test is of value in determining the presence of mucus in membranous specimens from mucous colic. The tests otherwise are rarely required.

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[1] N. Y. Med. Jour., Dec. 7, 1907.

*Albumin.*—Treat the feces with water slightly acidified with acetic acid. Filter the watery extract and employ boiling test as for albumin in the urine. Normally, *no albumin is present*, but it has been found in typhoid, occasionally in acute enteritis, and in chlorosis.

*Propeptone and Peptone.*—After the test for albumin has proved negative, the watery extract of the feces is treated with phosphotungstic acid, the precipitate is diluted with water and sodium hydrate, and a small amount of a weak solution of sulphate of copper added. A purple red (biuret reaction) shows the presence of both propeptones and peptones. To determine the presence of peptones separately, first precipitate the propeptones by ammonium sulphate in large amount.

Pathologically, peptone is found in typhoid, dysentery, tuberculous ulcer of the intestine, and in perforative peritonitis. Normally it is not present.

*Carbohydrates.*—The feces are first subjected to distillation. The residue is extracted with alcohol and ether; the extract boiled with water, filtered, and again boiled, with the addition of dilute sulphuric acid. Trommer's or Nylander's test is then employed.

*Examination for Starch.*—The watery extract of feces is examined with Lugol's solution, the presence of starch producing a blue color.

*For Sugar.*—A watery extract of feces can be directly tested by Fehling's method.

Normally, neither starch nor sugar are found.

*Gas Fermentation.*—Schmidt's method will be described later.

*Fat.*—The feces are treated with considerable ether, and the latter is separated and evaporated in a water-bath. The neutral fat, if present, remains visible.

To show the presence of soaps which do not dissolve in ether, another portion of fecal matter is first treated with acids which split up the soaps, and then extracted with ether; quantitative determination is complicated.

Normally, fat is never present macroscopically in the stools unless after ingestion of very large quantities. It may then be visible in very small portions, the size of a pea. Pathologically, fat may exist in large quantities in the fecal matter and give the grayish-silver fatty stools, especially in disease of the pancreas and whenever lymphatic absorption is disturbed.

*Blood.*—Fresh blood can often be recognized macroscopically. The tests for occult (concealed) blood are of importance. The best methods are the benzidin test (the latest); Weber's modification of the guaiac test; and the aloin test. These are fully described under Tests for Occult Blood in the Stomach-contents and Stools in Part II of this volume. Neither meat nor iron preparations should be ingested for two to three days previous to the tests. The hemin test has been employed. A small particle of fecal material is dried, powdered, and placed on a slide. A trace of sodium chlorid is added and a drop of glacial acetic acid poured on and thoroughly mixed. A cover-glass is placed over the specimen and the slide slowly heated. After cooling, a microscopic examination is made. In the presence of blood, hematin crystals are found (Fig. 211). These are reddish pink and rhomboid in shape.

*Bile-pigment.* — Normally, no unchanged bile-pigment is found in the feces. In catarrh of the small intestine it has been detected. The presence of bile-pigment is determined as follows: A particle of the colored fecal matter is brought into contact with a drop of fuming nitric acid. The yellow color passes through the various colors of the spectrum, red, violet, to green; in some cases green appears at once; or liquid feces can be filtered through filter-paper or a watery mixture can be made and then filtered. The paper is then dried and a drop or two of the fuming nitric acid poured on it. The colors will appear in rings if bile is present; or:

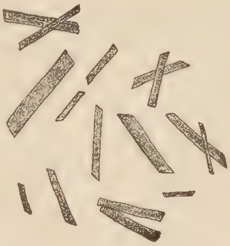
Fig. 211.—Hematin crystals.

A small quantity of the feces is treated with a concentrated watery solution of corrosive sublimate. Biliary pigments will turn the mixture green, or green appears in that portion where pigments are present.

*Biliary Acids.*—These usually accompany biliary pigments. They are revealed by Pettenkofer's test: A small quantity of feces is treated with alcohol and then the latter is evaporated. To the residue a weak watery solution of bicarbonate of soda is added, and to this mixture a small amount of cane-sugar and a few drops of sulphuric acid. Red or pink occurs when biliary acids are present.

*Urobilin.*—Normally, the biliary pigment in the intestinal tract becomes changed to *stercobilin*, which gives the brown color to the feces.

A small piece of fecal matter is treated with a concentrated watery solution of corrosive sublimate and thoroughly mixed with a glass rod. Urobilin (stercobilin) gives rise to a pinkish-red color; bilirubin, to a green color. Urobilin is normally present and is absent in pathologic conditions, while bilirubin is present in the latter.

*Fleischer's Test.*—Place a small quantity of feces in a test-tube with a small amount of alcohol to which has been added a few drops of hydrochloric or acetic acid. After a short time urobilin produces a yellow or brown color. If the alcohol is then poured off and a few drops of sodium hydroxid with a small quantity of zinc chlorid are added, there appears a green fluorescence in direct rays of light, and in transmitted light, pink or yellowish red, greater or less in proportion.

**Acholic and Colorless Stools.**—The acholic stool presents a grayish-white, ash-gray, or clay color due to absence of bile-pigment. The dejecta are of penetrating odor, buttery consistency, and on chemic and microscopic examination are found to contain much fat. The latter is present as needle-shaped crystals, or in sheaves of crystals, or, less generally, in fat-droplets. This type of stool occurs in conditions such as occlusion of the bile-duct, when there is an exclusion of bile from the intestine.

Stools may be entirely *devoid of color* or of a grayish-white color resembling true acholic stools, though there is *no jaundice or occlusion of the bile-ducts*. These stools are less putrid in odor and more acid. They contain enormous amounts of fat, like the true acholic stool, and *urobilin has been demonstrated in them*. Such movements occur in conditions when the absorption of fat is impaired, as in tuberculosis of the intestines and peritoneum. At other times abnormal quantities of fat are not present, and the lack of color is imputed to the presence of a colorless decomposition product of bilirubin, the leuko-urobilin of Mencki.

The conclusion that a stool contains an excessive amount of fat because it is apparently acholic is not justifiable unless a microscopic examination is made. An apparently acholic stool may also be due to excessive fat ingestion and urobilin be present.

**Fatty Stools (Steatorrhea).**—This term is applied to all cases in which isolated masses of fat are present in the feces and can be *recognized with the naked eye*. It appears in whitish or grayish lumps, or it may cling around the feces or be adherent to the vessel.

Ingestion of excessive fat even under normal conditions may produce an evacuation of superfluous fat. If the mucosa of the small intestine and the lymphatic system (mesenteric glands) lose their powers of absorption, fat must appear in the stools, as in tuberculosis of the small intestine, chronic tubercular peritonitis, intestinal catarrh, etc.

In occlusion of bile from the intestines with acholic stools, the fat is revealed microscopically and by chemic analysis, but fatty stools are not then spoken of in the clinical sense.

Steatorrhea is not *per se* diagnostic of pancreatic disease. In the absence of icterus and of demonstrable intestinal disease, fatty stool is probably due to pancreatic disease. Disturbed digestion of fat is diagnostic of pancreatic disease. Müller shows that qualitative changes in the fat (lipolysis) is much slighter, only 39.8 per cent. in pancreatic disease, where it is 84 per cent. in healthy subjects, or even in those with icterus, if the pancreatic juice has free access.

**Ferments.**—A glycerin extract can be made of the feces, or the fecal matter may be mixed with water containing a small proportion of thymol and filtered.

To test for *trypsin*, the filtration extract is made alkaline by the addition of soda bicarbonate and a few flakes of fibrin added. The solutions are kept at a blood temperature for a few hours and then tested with potassium hydroxid and a weak solution of copper sulphate. If trypsin is present, there will be a pinkish-red reaction (biuret) in consequence of peptone.

*For Diastase.*—A few cubic centimeters of the filtrate are mixed with about one-half the amount of a starch solution and kept at a blood temperature for about thirty minutes. The mixture is then subjected to Fehling's or Trommer's test. Normally, these ferments are absent,[1] but in pathologic conditions, especially in diarrhea, they are frequently found. Wynhausen[2] describes a new test for the pancreatic activity. (See Testing the Pancreatic Functions.)

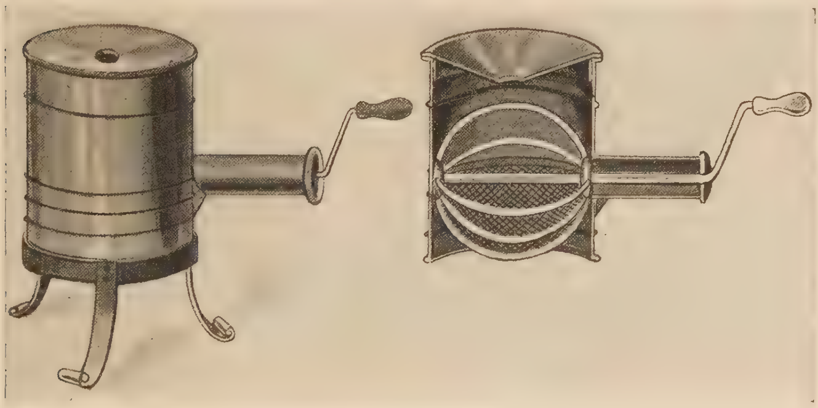

Fig. 212.—Einhorn's stool sieve.

**Concretions, Foreign Bodies, Worms.**—To examine for such material the feces should be thoroughly mixed with water and poured through a sieve. This can be improvised—a circular wire rim can be fitted to a water-closet seat, and attached to the wire a network bag made of two thicknesses of cheese-cloth, practically a dip-net. The patient defecates in the net, and water can then be poured through several times until only the more solid parts remain.

Boas has constructed a stool sieve, and Einhorn's, as in Fig. 212, is readily understood. Water is poured through and there is a stirring apparatus.

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[1] Goldschmidt (Deut. med. Wochens., 1909, No. 12, xxv, 522) and Gross (Ibid., 1909, No. 16, xxv, 706), using the latter's method for detecting trypsin, have discovered it in the feces of all normal persons examined. This, if confirmed, will prove a great advance.

[2] Berl. klin. Wochens., July 26, 1909; also Med. Rec., Sept. 11, 1909.

*Concretions.*—Among such are gall-stones, pancreatic calculi, enteroliths, and coproliths. Biliary calculi are readily recognized when of any size. The principal constituents are cholesterin, bile-pigment, and lime.

Tests for small biliary concretions (sand) are as follows: first, powder 30 grains (2.0) of the mass and treat with ether, 5 drams (20 c.c.), mix and filter, evaporate, and test for cholesterin. Dissolve part of residue in hot alcohol and allow it to evaporate. Microscopic examination of the precipitate shows rhomboid crystals with ragged edge (cholesterin); second, another part of the residue is placed on a slide, a drop of concentrated sulphuric acid added and covered with a cover-glass, the cholesterin crystals become carmine at their edges, add 1 drop of Lugol's solution and a violet color arises; finally, a portion of the residue is treated with hydrochloric acid, a trace of iron chlorid, and then evaporated. If cholesterin is present, a blue color occurs.

The residue of the original ether mixture is treated with dilute hydrochloric acid mixture, heated, and extracted with chloroform after cooling. The chloroform extract is tested with Gmelin's reaction (fuming nitric acid). Bile-pigment produces the rainbow play of colors.

*Pancreatic Calculi.*—These usually have a rough surface, are brittle, and may be faceted. They are soluble in chloroform, and on evaporation produce an aromatic odor.[1] Bile-pigment and cholesterin are usually absent. (See Pancreatic Lithiasis.)

*Enteroliths.*—Calculi formed in the small intestine consist of inorganic salts (lime and magnesia). They are light in color and usually of small size. They form occasionally after the extensive use of lime and magnesia. Rarely they cause obstruction.

*Coproliths* (*fecal calculi*) are found in the large bowel, chiefly where there is retardation to the passage of feces, as in the cecum, appendix, sacculi of the colon, sigmoid, and rectum. They are of stony hardness and sausage shape and show concentric rings on section. They may attain considerable size and even cause intestinal obstruction.

*Foreign Bodies.*—Bodies which have been swallowed may pass through the entire bowel and be passed in the feces, such as bones, coins, marbles, needles, etc. Concretions of shellac have been found in the stools of patients who have drunk furniture polish. Hair-balls may be found.

**Microscopic Examination.**—The microscopic examination of the feces is often of great assistance to diagnosis. For examination for amebæ, the stool should be kept warm. A *thermos bottle is useful for this purpose.*

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[1] Minch, Berl. klin. Wochens., 1898, No. 8.

To diminish the disagreeable odor of a watery stool, place it in a conic glass and cover it with a layer of ether. If it is mushy or firm, it can be spread on a plate and covered with a layer of spirits of turpentine, or a 5 per cent. solution of carbolic acid or thymol, or 4 per cent. formalin.

Diarrheal stools may be examined without further preparation.

With solid fecal matter, a small piece of feces may be placed on a slide and mixed with a drop or two of normal salt solution. If there is odor, a 1 per cent. formalin solution may be added. The findings depend on the diet. With meat diet, no vegetable residue is found, and vice versâ. With a mixed diet, in a normal stool, there will be plant cells, the remnants of various vegetables and fruits, no starch granules, meat-fibers changed beyond recognition, or with slight striation (Fig. 213). More commonly they appear as oval yellow translucent masses with a high degree of refractibility. The presence of numerous meat-fibers striated and with nuclei is pathologic, showing deficient pancreatic digestion (tryptic). They may appear as spirals (Fig. 214).

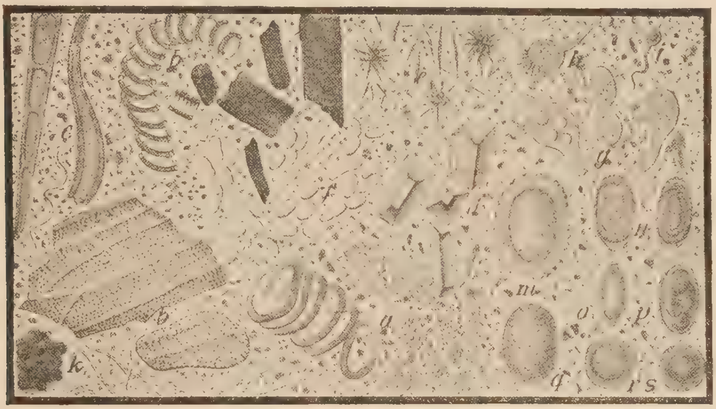

Fig. 213.—General view of the feces: *a*, Epithelial cells and leukocytes; *b*, stone-cells; *c*, cuticular formations; *d*, crystals of ammoniomagnesium phosphate; *e*, fat-crystals; *f*, yeast-fungi; *g*, Amœba coli; *h*, Trichomonas intestinalis; *i*, Cercomonas intestinalis; *m*, ovum of ascaris; *n*, ovum of oxyuris; *o*, ovum of trichocephalus; *p*, ovum of ankylostomum; *q*, ovum of bothriocephalus; *r*, ovum of Tænia saginata; *s*, ovum of Tænia solium (Jakob).

*Fat.*—Fat appears microscopically as colorless small globules or as needle-shaped crystals (fatty acids) or in sheaves (soaps). The fatty acids disappear when heated or when ether is added; soaps remain unchanged. Sudan dye-stuff, in concentrated alcoholic solution, stains plain fat bright red, while crystals of fatty acid and the soaps remain unchanged. In pathologic conditions these forms of fat are markedly increased, as in affections of the liver, pancreas, and intestines. Normally, they are scanty.

*Crystals.*—Oxalate of lime, calcium carbonate, neutral phosphate of calcium, and ammonium magnesium phosphate are found in normal as well as pathologic feces and have no diagnostic importance. Bismuth, if administered, occurs as dark brown or nearly black rhomboid crystals. Hematoidin appears in rhombic crystals of orange or red color, shortly after intestinal hemorrhage. Charcot-Leyden crystals (Fig. 215) are fine colorless, pointed octahedra. These when present excite the suspicion of helminthiasis (intestinal parasites), and their persistence after removal of the tenia shows the

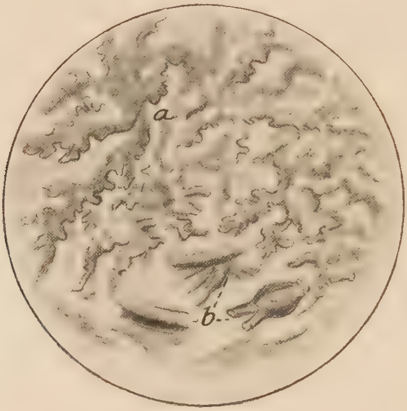

Fig. 214.—*a*, Spirals of undigested meat-fibers in stool; *b*, pieces of bronchi.

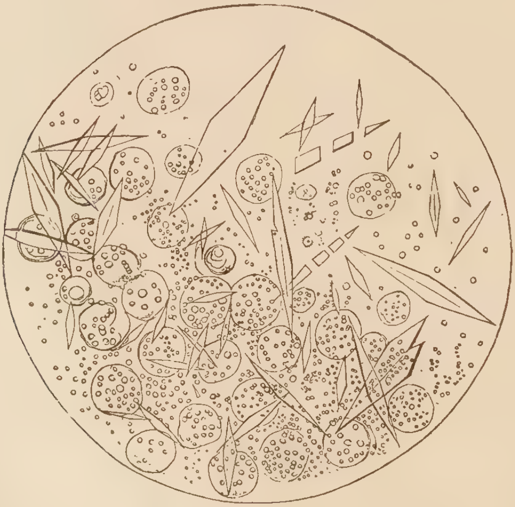

Fig. 215.—Charcot-Leyden crystals (after Riegel).

head has probably not been removed. They occur occasionally in normal stools and in typhoid, dysentery, and phthisis.

*Epithelium.*—Epithelial cells when present in large numbers always indicate an inflammatory condition of some part of the intestinal tract. Cylindric epithelial cells are found in abundance in inflammation of the intestinal mucosa (Fig. 216). They cause the cloudy appearance of the mucus. If bile-stained specimens of epithelia are met with, the small intestine is involved. Degenerative forms without nuclei are mostly seen, though well-preserved cylindric or goblet-cells are often found.

*Red blood cells* are rarely observed unless hemorrhage is from the colon or rectum, as in dysentery. Hemorrhage higher up gives a brownish-red color to the feces, and hematoidin rhombic crystals in some cases, and the blood cells cannot be recognized microscopically.

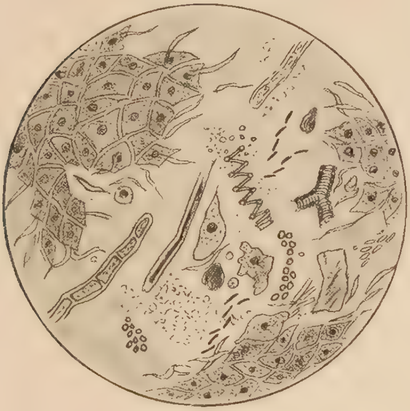

Fig. 216.—Chronic intestinal catarrh: Groups of epithelial cells, detritus, some muscle cells partly digested, bacteria, plant cells, and yeast cells.

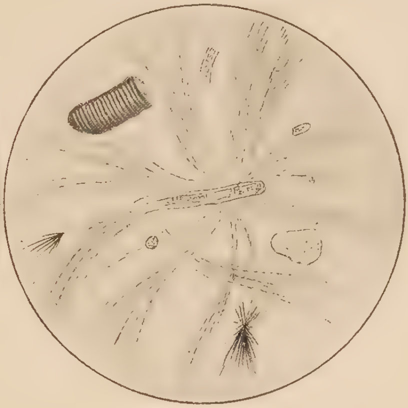

Fig. 217.—Intestinal catarrh: Considerable mucus, some plant cells, muscle cells, and fat crystals.

*Pus* occurs in the dejecta in ulcerative processes when an abscess communicates with the bowel, or in dysentery, or in any form of intestinal ulcer. It presents the usual characteristics.

*Mucus* when bile-stained indicates disease of the small intestine; and if colorless, catarrh of the large intestine or lower part of small intestine is present. Mucus also occurs with mucous colic, in which condition no catarrh exists. Mucus is thread-like in appearance, though occasionally amorphous (Fig. 217). Hyaline particles of vegetable residue must not be mistaken for mucus. Iodin stains it blue. Thionin colors it reddish violet and other proteins blue. Mucus has no definite structure.

*Tumor.*—Fragments of tumor may rarely be found in the feces and its character determined by the microscope.

*Microöragnisms.*—A large portion of the stool is constituted by bacteria, as already mentioned. Among the most important are the Bacillus coli, Bacillus lactis aërogenes, Bacillus bifidus, Bacillus aërogenes capsulatus (gas-forming), and Bacillus putrificus.

The Bacillus coli is of importance in reference to the indolic type, and the Bacillus aërogenes capsulatus, to the saccharobutyric type of intestinal putrefaction. The Bacillus lactis aërogenes causes fermentation of milk and the production of lactic acid.

The lactic-acid-producing bacilli are held to be antagonistic to putrefactive changes. For a description the reader is referred to any work on Bacteriology. Typhoid, tubercle, dysenteric, and the cholera bacilli are the chief pathogenic micro-organisms found in the feces which are of interest to us.

## TESTING THE INTESTINAL FUNCTIONS

Boas has obtained intestinal juice by passing the stomach-tube into the empty organ and massaging the region of the liver, thus forcing the juice into the stomach. Hemmeter and Kuhn have passed the tube directly. These procedures are uncertain and possess no advantages.

Einhorn[1] has devised a new method of obtaining the intestinal juice by means of a duodenal bucket. The quantity secured (and it is not always obtained) is infinitesimally small; there is the possibility of an admixture of gastric juice and saliva during withdrawal, and the procedure requires three to five hours or even longer. It does not admit of the thorough technic as secured by Schmidt's test-diet.

Einhorn has recently suggested passing a soft tube along the duodenal bucket cord (on the principle of Gouley's tunnel sound along the filiform bougie), and then aspirating the intestinal contents with a bulb.

Einhorn's bead test[2] the author does not believe to be sufficiently accurate. Testing the motor function with foreign bodies (beads) is not a proper criterion; the food material attached to each bead is too infinitesimally small in amount to test the digestive capacity for an average diet; it may work loose from the beads, in which event it could not be recovered and wrong deductions might result. There is a slight element of risk from fish-bone escaping from the bead and damaging the mucosa. Additional methods of testing the secretion of the pancreas are described under Diseases of the Pancreas.

The author believes *the stomach functions should be tested separately*, and one should not depend on the connective tissue test for the stomach test also, as suggested by Schmidt.

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[1] N. Y. Med. Jour., June 20, 1908.

[2] Med. Rec., Feb. 10, 1906; Ibid., Oct. 26, 1907; Jour. Amer. Med. Assoc., Feb. 2, 1907; Therapeutic Gaz., Jan. 15, 1908.

Boldyreff's oil test-meal, the tests of Mett, Volhard, and Gross, and the duodenal pumps of Gross and Einhorn are described under Diseases of the Pancreas.

To E. E. Smith, of New York, I am indebted for a description of the technic—the one I recommend to the general practitioner.

**Tests of the Intestinal Functions.**—The determination of the functional activity of the stomach is made with relative ease, compared to the similar examination of the intestines. Correspondingly, methods for such determination have long been applied to the former organ, while they have recently found application to the latter; and even now the diagnostic determination of intestinal function by exact methods is only in its infancy. The problems presented are essentially these:

(*a*) Is intestinal secretion, including pancreatic and biliary secretions, normal in quality and quantity? (*b*) Is intestinal absorption normal? (*c*) Is the intestinal motor activity normal? If not, in what respect is each of these processes abnormal?

The investigation of these problems is made by the use of a test-diet. While in the case of the stomach the diet is relatively simple and the digestive mixture withdrawn for examination within a few hours, in the case of the intestines only inferences as to the intestinal contents may be reached, and then indirectly from the conditions affecting the bowel contents after they have been ejected, perhaps in one or several days. The feces corresponding to the test-diet may be indicated by a material administered for the purpose, usually soot in capsules, No. 00 hard gelatin capsules filled with soot accomplishing this purpose; or carmin may be used, 5 grains (0.3 gram) in a capsule. Either of these is administered at the beginning of the first meal of the test-diet. The subsequent appearance of a black or red stool indicates that the intestinal contents corresponding to the special diet are being ejected.

The administration of a special substance to mark the stools is, in a majority of instances, not absolutely necessary, the diet being of a nature that gives rise to a stool sufficiently distinctive for recognition. Until the observer is familiar with the examination, it is advisable to employ one of the the substances mentioned.

The test-diet to be employed is selected to meet the requirement that it shall present a sufficient quantity of *all classes of food stuffs to test the digestive capacity*, that the digestive processes shall not be unduly anticipated in the preparation of the food, and that very little food residue shall be present in the ejected bowel contents. Naturally, the stomach plays its usual part, so that the test is not limited to the intestinal tract proper, but applies to alimentation as a whole.

The test-diet made use of is a modification of the original Schmidt-Strassburger diets. Instead of three diets, as were originally employed in connection with the fermentation test of these observers, one diet is now advocated which presents the conditions essential for the meat test. This diet, as recently described by Schmidt, is as follows:

*In the Morning.*—0.5 liter milk, or, if milk does not agree, 0.5 liter cocoa (prepared from 20 gm. cocoa powder, 10 gm. sugar, 400 gm. water, and 100 gm. milk), to this add 50 gm. zwieback.
*In the Forenoon.*—0.5 liter oatmeal gruel—made from 40 gm. oatmeal, 10 gm. butter, 100 gm. milk, 300 gm. water, 1 egg—(strained).
*At Noon.*—125 gm. chopped beef (raw weight), broiled rare with 20 gm. of butter, so that the interior will remain raw; to this add 250 gm. potato broth (made of 190 gm. mashed potatoes, 100 gm. milk, and 10 gm. butter).
*In the Afternoon.*—As in the morning.
*In the Evening.*—As in the forenoon.

This diet consists of:

1.5 liters milk,
100 gm. zwieback,
2 eggs,
50 gm. butter,
125 gm. beef,
190 gm. potatoes and gruel of
80 gm. oatmeal.

It contains about:

102 gm. albumin,
111 gm. fat,
191 gm. carbohydrates, or a total of 2234 calories (raw calories).

The test is generally given for three days, sometimes longer; at any rate until a stool is obtained, which comes with certainty from this diet.

Steele advocates an arrangement of the diet to conform to American dietary habits, which still maintains the essential features of the above. It consists of:

2¾ pints milk,
3 ounces well-dried toast,
2 eggs,
1½ ounces butter,
¼ pound tender rare steak,
6 ounces mashed boiled white potato, and gruel made from
2½ ounces ordinary oatmeal,
½ ounce sugar.

This may be given somewhat as follows:

*Breakfast.*—2 eggs, one-third of the amount of toast and butter, 2 glasses of milk, oatmeal, and sugar.
*Dinner.*—The steak and potato, one-third of the amount of toast and butter, 1½ glasses of milk.
*Supper.*—2 glasses of milk, remainder of toast and butter.

For the collection of the stools, where they are not to be transported for any considerable distance, an ordinary tin basin of 24-ounce capacity serves well for a receptacle, as suggested by Prof. L. B.

Mendel. This may be supplied with a cover, consisting of a cake tin of appropriate size; or, where this is not readily provided, a small pie tin may be used. This outfit is inexpensive (8 cents), adequate, and where a considerable number are to be kept, may be advantageously stacked. Moreover, where the stools are to be weighed and dried for exact quantitative analysis, this may be directly done in the weighed basins, thus avoiding the transfer of the specimens.

If the feces are to be transported for any considerable distance, a pint glass jar (with patent air-tight top) serves admirably for the collection. The specimen may be hermetically sealed. If transported in ordinary wide-mouthed bottles, the stoppers should be tied on, as gas formation is quite likely to produce sufficient pressure to force out any stopper not securely fastened.

The characteristics of the test-diet stool, aside from the coloration given by the special marking substance administered, are the light brown color and uniform consistency. It usually appears at the second or third defecation after the beginning of the test-diet.

The *period of time required* for the passage of food through the entire alimentary tract is of importance and is readily observed. Normally it takes about twenty-four hours. It is not necessarily, though it is commonly, related to the frequency of defecation. In some cases the stool appears with regularity and is fairly copious, yet the patient suffers from fecal accumulation—a latent constipation. The period of passage may suggest the seat of the intestinal disturbance in diarrhea, since it is only decidedly increased when the cause is high up. Strauss has shown that chronic colitis may be accompanied by several watery movements a day with a normal period of passage. The examination of the collected stool should be made while the feces are perfectly fresh.

*Macroscopic Examination.—This is the most important part of the procedure.* Experience *with this part of the investigation* may enable the observer *to at once recognize some defect in alimentation.*

Note the consistence, color, and odor. Inspect the surface of the formed stool for morbid products, notably for pus, blood, and mucus, which are to be removed for microscopic and bacteriologic examination. Bits of tissue from some diseased area may also be sought for, but their occurrence is so unusual that it is exceptional when they are found.

The mass of feces is then well mixed, for which procedure a wooden or tin-plate spoon is useful; and a piece the size of a walnut transferred to a mortar, in which it is thoroughly but not forcibly ground with distilled water added gradually until the whole is of a uniform fluid consistency and no small masses of fecal matter remain. The fluid feces is then examined in thin layers, conveniently in a Petri or similar larger dish, against a black background, with the naked eye or low-power magnifying glass, for all elements that may be differentiated. In normal digestion only a few brown points smaller than pin-heads will appear, these consisting of chaffy remains of oatmeal gruel and remains of cocoa, if this latter has been taken. Under pathologic conditions, there may be present:

1. Mucus, which appears usually as larger or smaller, soft, glossy, translucent flakes, often bile stained; infrequently, when from the large intestine, white or brown, with a gummy or almost leathery hardness.

2. Pus, blood, parasites, stones, and other foreign bodies.

3. Connective tissue and tendons, distinguished by their whitish-yellow color, thread-like appearance, and solid consistence.

4. Muscular tissue, chiefly in very small, brown colored rods, like splinters of wood.

5. Potato, appearing like boiled tapioca grains, readily confused with flakes of mucus. The distinction is made with the microscope.

6. Large crystals of ammoniomagnesium phosphate, which grate when the specimen is ground.

*Microscopic Examination.*—This serves chiefly to complete the gross inspection. In addition to the preparations of material selected during the macroscopic examination, three slide preparations are made. The *first* consists merely of a drop of the liquefied stool under the cover-glass. The *second*, a drop of the liquefied stool mixed with a drop of acetic acid, heated to the beginning of boiling and covered with a cover-glass. The *third* consists of a drop of the liquefied stool mixed with potassium iodid solution of iodin and covered with a cover-glass.

Inspection of the first slide preparation by the aid of the microscope, using a high, dry lens, reveals finely divided material consisting of bacteria and mostly unrecognizable detritus, in which are imbedded:

(*a*) Isolated fragments of muscle-fibers, usually bile stained, partially digested, but occasionally with the transverse striations recognizable

(*b*) Larger or smaller yellow crystals of the alkali earth salts of the fatty acids.

(*c*) Colorless soaps.

(*d*) Isolated potato cells, without distinguishable contents.

(*e*) Particles of oatmeal and cocoa shells, where the latter is taken instead of milk.

The second slide gives a general idea of the fat present in the stool. While hot, the fatty acids, liberated in the acetic acid, appear as drops; on cooling, these congeal to small needle-like crystals.

In the third preparation potato remains have a violet color, while isolated fungous spores (Clostridium butyricum) may appear blue.

The pathologic findings which the slide may present, in addition to those enumerated under the macroscopic examination, are:

*Slide I.*—Muscle fragments in greater number and better state of preservation, particularly with retained nuclei; drops of neutral fat; needles of fatty acids and soaps; many groups of potato cells.

*Slide II.*—Massive fatty acid drops and crystals.

*Slide III.*—Blue starch grains, free or in the potato cells; oatmeal cells; any considerable number of blue-staining fungous spores or thread-like bacteria.

*Bacteriologic Examination.*—The recognition of the tubercle typhoid, Shiga, or cholera bacillus calls for technic which will be found described in special treatises. The selection of material to be examined for tubercle bacilli is best made from the surface of formed stools, since in soft movements morbid products from the diseased area will be so mixed with the feces as to easily escape detection.

For amebæ a saline cathartic such as magnesium sulphate should be given and the warm liquid stool examined. The stool may be kept warm in a Thermos bottle for transportation to the laboratory. Mention has already been made of the detection of bacteria and fungi which are colored blue by iodin (granulose reaction).

Procedures that yield information of considerable value in the less specific forms of intestinal infection have recently been advocated by Herter. Of first importance is the preparation of smears of the mixed stools on microscopic slides, stained by the Gram method. The relative number of Gram-positive bacteria, as also their character, is of diagnostic value, since they are relatively few in health and in meat-free diet, while they are increased in some diseased conditions and when notable quantities of meat are eaten. Not only is it of value to note the actual increase, but the potential. The latter is determined by the observation of the relative number of Gram-positive bacteria in the residues in the sugar-bouillon tubes (see below), a predominance of Gram-positive bacteria indicating a pathologic tendency of the fecal flora.

The sugar-bouillon tubes just mentioned are ordinary fermentation tubes, containing 1 per cent. lactose-bouillon, 1 per cent. glucose-bouillon, and 1 per cent. saccharose-bouillon respectively.

Normally, little fermentation occurs when the tubes have been incubated at 37° C. for twenty to twenty-two hours. Active gas production, so that it accumulates to the extent of more than one-third of the tube capacity, is most frequently due to the predominance of a bacillus identical with or allied to the Gram-positive Bacillus aërogenes capsulatus, the growth of which replaces the Bacillus coli communis, the normal intestinal inhabitant.

*Chemic Examination.*—The *chemic reaction* is best determined by smearing one side only of moistened red and blue litmus-paper with the diluted (see Macroscopic Examination) fecal matter. After some time the reaction is noted on the opposite side. It is usually amphoteric, feebly acid, or alkaline.

*Excretion of Bile.*—The *sublimate test* for unchanged bile-pigment is performed by adding some of the diluted fecal matter to a considerable excess of strong solution of bichlorid of mercury, allowing the mixture to stand over night. The normal *feces are colored red;* more intensely, the fresher and less decomposed the excrement. Herter points out that *this red coloration* may be pathologically increased in excessive saccharobutyric putrefaction. This excessively strong reaction is probably due to the reduction of the bilirubin to hydrobilirubin through the Bacillus aërogenes capsulatus. *In the presence of bilirubin, a green coloration is produced.* This, even to the extent of microscopically small particles, is pathologic. *A negative sublimate test suggests suppression of bile.* This last is of value, as it indicates that an acholic stool is bile free and that the colorless stool is due to complete occlusion of bile from the intestines. An incomplete test with fresh stools shows abnormal processes of decomposition.

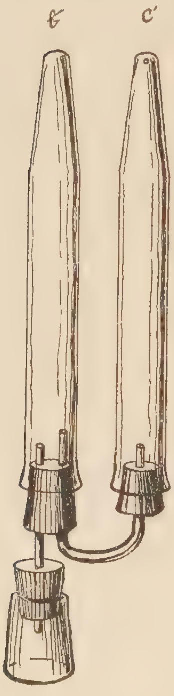

Fig. 218.—Strassburger's tube.

*Functions of the Liver.*—The best method of testing the functions of the liver depends on the power of the normal liver to metabolize 100 grams of *levulose* when given all at once in solution, as in weak coffee, on an *empty stomach.* Normally, no trace of this should appear in the urine by Fehling's test. The urine should be first examined six hours after its ingestion.

The *fermentation test* of Schmidt and Strassburger for fermentable carbohydrate or putrescible protein is performed in the Strassburger fermentation tube (Fig. 218).

A 5-gram portion of the well-mixed, undiluted, fresh excrement, or proportionally more of the thinner material, is well mixed with sterile water; the chemic reaction noted; and then the mixture is introduced into the lower vessel of the apparatus.

The bottle is entirely filled with water and stoppered, with exclusion of air. The adjacent upper tube is filled or nearly filled with water, while the distal tube remains empty. The apparatus is incubated at 37° C. for twenty-four hours.

The extent of gas formation is indicated by the amount of water displaced by the gas from *b′* and which accumulates in the distal tube *c′*. Normally, there is practically no gas formation, and the chemic reaction of the fecal mixture remains about unchanged. Gas production to such an extent as to introduce an amount of water into the distal tube equal to one-third its capacity is pathologic. If coincidently with the gas production the chemic reaction has developed a decidedly increased acidity, the gas production is due to carbohydrate fermentation; if alkalinity, albuminous putrefaction has occurred. When the proximal tube is opened it gives off a butyric acid odor in the former case and a putrefactive odor in the latter. The color of fermenting feces is generally brighter; of putrefying feces, darker. The test is more especially applicable to the test-diet stools.

If the condition approximates the normal, a further test should be carried out for accuracy. In such event, the patient is placed on a diet which differs only from the first in the absence of meat and potato. If there is still a positive result, the diagnosis of "fermentative dyspepsia" is justifiable.

*Putrefactive Products.*—Tests for indol and skatol may be applied to the distillate, using 10 gm. of the feces mixed with 120 c.c. of water, and the whole made alkaline, a bit of paraffin added to prevent frothing, and the first 50 c.c. collected.

A suitable apparatus for conducting the process consists of a 500-c.c. capacity long-neck Kjeldahl digestion flask connected with a Liebig condenser. Distillation with steam is sometimes advantageous; 10 c.c. of the distillate is rendered slightly alkaline with sodium or potassium hydroxid and an excess of a fresh solution of beta-naphthoquinone-sodium-monosulphonate added. The substance, in the course of a few minutes, reacts almost completely with the indol present, but not with the skatol; with the resulting formation of a bluish precipitate with much indol, and a mere coloration of the solution with little. If more than a trace is present, the reaction is conducted with the remaining 40 c.c. of the distillate, the indol-naphthaquinone compound removed from the whole by filtration, and from the portion remaining in solution by distillation, after acidifying.

The distillate containing the skatol, if necessary, freed from indol as described, is tested by the use of a well-marked excess of dimethyl-amido-benzaldehyd (Ehrlich's aldehyd), being boiled with a 5 per cent. solution in 10 per cent. sulphuric acid. Dilute hydrochloric acid is added to the point of the production of the maximum color intensity and the mixture rapidly cooled. The presence of skatol is indicated by the blue coloration. The color may with advantage be extracted with chloroform.

If the process described is to be conducted quantitatively, 25 grams of feces should be employed, and distillation continued till the distillate is free from substances reacting with the above reagents, the color shaken out with known volumes of chloroform, and the depth of color compared by the aid of the Duboscq colorimeter, with a similar chloroform extract obtained by starting with solutions of known strength of indol and skatol respectively.

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Schmidt, A., translated by C. D. Aaron, The Test-diet in Intestinal Diseases, vol. lxxvii, 1906.
Steele, J. Dutton, Medical News, 1905, p. 1158, vol. lxxvii.

**Results.**—In interpretation of results, we have to consider both the occurrence of pathologic admixtures and an increase of the constituents from the test-diet.

A thin coating of mucus is normally collected by the hardened fecal matter. Otherwise the appearance of this product indicates catarrh of the mucosa, the mucus containing many cellular elements. An exception is the overproduction of mucus in colica mucosa, with cellular elements less abundant and with the striking consistence.

Pus, blood, masses of epithelia from the mucosa, and similar elements carry the pathologic significance of these products, and require investigation and interpretation, as when found elsewhere.

Considering an increase of the constituents of the test-diet, a distinct connective-tissue increase indicates deficient gastric[1] digestion. An excessive quantity of meat-fibers points to deficient protein digestion in the small intestine. The albumin fermentation (putrefactive) test further indicates increased protein in stools, frequently derived from pathologic secretions, probably associated with an abnormal putrefactive flora. The bacteriologic examination, especially as to fermenting and Gram-positive bacteria, is of value along these lines. Also tests for putrefactive products, if increased activity in this direction in the stool itself is to be investigated.

Starch granules, revealed microscopically, show deficient starch digestion in the intestines, due either to rapid passage of the contents or disturbed secretion. Defective carbohydrate digestion is also indicated by an abnormal carbohydrate fermentation test.

Only a considerable increase of fat is pathologic. Such a stool is indicated by the quantity of the stool itself, the light (whitish) color, and a marked acid reaction, as well as by the microscopic findings. A deficient flow of bile or of pancreatic secretion is the usual cause; in the former case the stool not containing pigment (sublimate test); in the latter showing an associated increase of meat-fibers, frequently with retained nuclei. Functional disturbances of fat digestion are said to occur, but other possibilities must be excluded in reaching this diagnosis.

## MECHANICAL PROCEDURES

**Direct Lavage of the Duodenum.**—This method has been suggested by Gross,[2] by the employment of his duodenal tube. Siphonage is employed as in lavage. The author finds the method impractical and does not recommend it.

**The Enema; Intestinal Irrigation; Proctoclysis.**—For injection into or irrigation of the intestines there are *four methods*, all of which have their special applications. They are:

1. The enema.
2. Irrigation with a single tube.

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[1] The author advocates a separate test of the gastric functions.

[2] N. Y. Med. Jour., Jan. 28, 1911.

3. Irrigation with a double-current tube or with two tubes.
4. Proctoclysis, the drop method of injection.

*Uses of irrigations or enemata* are as follows: The local treatment of diseased conditions, as of catarrhal colitis.

The relief of congestion or acute inflammation, as of the rectum or prostate.

The relief of pain and irritability, as in spasm of the sphincter.

The absorption of inflammatory products, as of postuterine adhesions.

To replace the loss of fluid in the body, as in cholera.

To dilute the poison of disease, as in uremia.

To increase the flow of blood to a part, as in insufficient menstruation.

To check hemorrhage (extreme cold or heat). Locally, as in bleeding ulcers of the rectum; in an adjacent organ, as in uterine hemorrhage.

Reflex effects through the sympathetic ganglia, on the circulatory apparatus, on the secretions, as a tonic stimulant, and the revulsive effects. On the circulatory apparatus, as in shock. On the secretions, as in the production of sweating, bowel action, and urinary secretion in uremia. As a tonic stimulant, as by use of the alternate hot and cold douche in diminution of erectile power. The revulsive effect, as the production of bowel action in apoplexy. The reflex effect on a distant organ, as from enemata in jaundice.

The effect on the heat centers—the temperature can be raised in shock by hot irrigation, or lowered in fever by cold irrigation or by enemata.

Simple cleanliness, removing undigested food products, and preventing auto-intoxication.

Antispasmodic, as by relieving spasm in colic.

Mechanical, as in intussusception.

Water as a vehicle—the nutritive enema.

*Physiologic Experiments.*—In a series of experiments at Columbia University some years ago the author demonstrated on animals (Fig. 219), and later clinically, that enteroclysis at 110° to 120° F. best stimulated the heart in shock; that renal secretion undergoes a double cycle of increase from enteroclysis at high temperatures (110° to 120° F.), both from intestinal absorption and from the increased blood flow through the kidney; while with lower temperatures the increase is merely from intestinal absorption.

Normal saline solution has a specific effect in increasing renal secretion. Cold irrigations first stimulate, then depress. Body and blood temperature are increased by hot irrigations and diminished by cold.

These experiments[1] were completely reported.

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[1] Enteroclysis, Hypodermoclysis, and Infusion, Kemp; Hydrotherapy, S. Baruch; Enteroclysis, Reference Hand-book of the Medical Sciences, 1900.

1. *Enema.*—The enema *may be high, with a colon-tube* (Fig. 220), which should be thoroughly lubricated and the water should flow while inserting. It should be administered with the patient on the left side, the dorsal position, with the hips elevated, can be employed, or, for high injection, in the knee-chest posture, when a high enema can thus be given with a short tip. I prefer never to give more than 1 to 1½ quarts (liters).

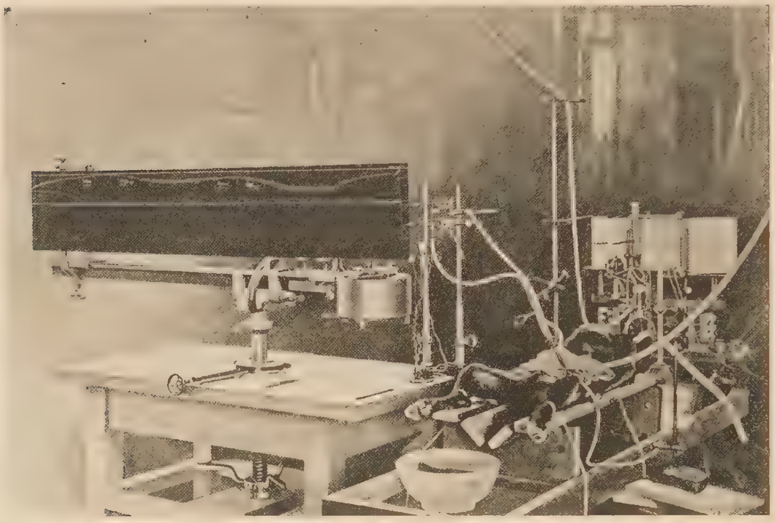

Fig. 219.—Method of performing physiologic experiments.

Milder medicated solutions can be employed in this way. The low enema is of more value for low impaction or to relieve local irritation in the rectum or adjacent organs.

2. *Irrigation with a Single Tube.*—There are four modifications of this method:

Fig. 220.—Colon-tube.

(*a*) A colon-tube is inserted into the bowel, a funnel attached, and by raising and lowering the funnel the bowel is washed out.

(*b*) A fountain syringe can be attached to the colon-tube, and when sufficient fluid has flowed in the fountain syringe the connection is detached and the fluid flows out through the colon-tube.

(*c*) The patient can void the fluid around the colon-tube or catheter during irrigation. This is the method usually employed with infants. Elmer Lee carried out this technic at the cholera stations (Fig. 221). An irrigating jar may be substituted for the rubber bag.

(*d*) A glass Y or T tube[1] is attached to the colon-tube, as in lavage of the stomach; one branch is connected with the fountain syringe, the other is to a soft carry-off tube. By alternately pinching the soft-rubber outflow and inflow tubes the bowel can be irrigated.

3. *Double-current Irrigation with Two Tubes or a Recurrent Tube.—Advantages.*—The quantity of the fluid is under the control of the operator, since it can be regulated by manipulation of the outflow and inflow tubes.

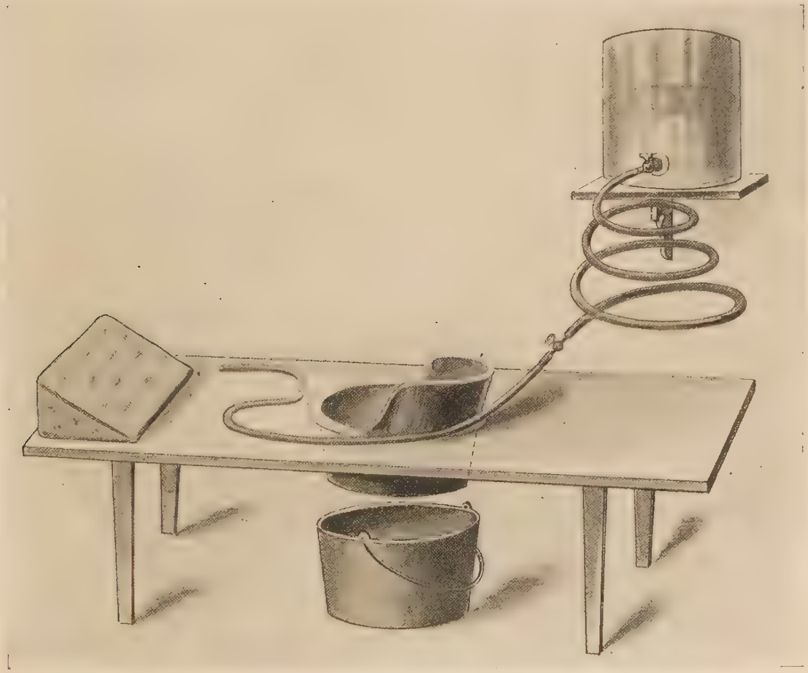

Fig. 221.—Lee's cholera table.

The labor is placed upon the operator and not upon the patient, and there is no straining to overcome the resistance of the sphincter. The straining of self-evacuation is avoided, and mere mechanical cleansing of the bowel is employed. The temperature of the fluid entering the bowel can be kept constant.

Tympanites is relieved best by this method, the return flow carries off the gas by suction. With the enema the gas frequently collects in the intestines behind the injection and it is often impossible to exert sufficient force to expel it with the enema.

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[1] Enteroclysis, Hypodermoclysis, and Infusion, Kemp; published by J. T. Dougherty, 1900.

Two catheters or two small rectal tubes passed through a perineal pad can be improvised for this purpose. The illustrations of the author's tubes are shown (Figs. 222–226). They are readily understood. J. P. Tuttle's, Hemmeter's, and various recurrent rectal tubes are described by the author in his manual " Enteroclysis." The hard-rubber tube with metal center is the best. All metal tubes are good for hospital work.

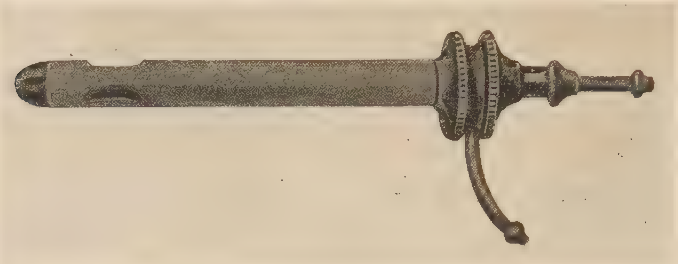

Fig. 222.—Kemp's flexible recurrent rectal irrigator.

The flexible tube is excellent for sensitive cases or for young children.

To the middle tube of the irrigator is attached the tube of the fountain syringe; to the curved tube the outflow soft-rubber tubing is fastened. This last must be pinched, as it is the larger, in order that fluid may pass up the bowel. By alternately pinching the inflow and outflow the quantity of fluid may be regulated.

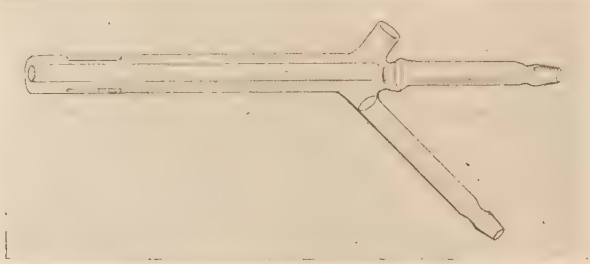

Fig. 223.—Kemp's glass rectal irrigator (recurrent). Cork opening above for cleansing.

The height[1] of the douche bag should be 3 to 5 feet above the patient.

Precautions before insertion of the tube: (1) Allow the irrigating fluid to flow from the tube, so as to force out all air and then check the flow. (2) As the tip of the instrument passes through the sphincter into the bowel, it is well to start the flow, so as to force the mucosa away from the irrigator and fenestræ. Also the entrance of the tube is not interfered with by the resistance of the mucous membrane.

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[1] Modern Methods of Intestinal Irrigation, The Medical Brief, Dec. 1910; also Manual on Enteroclysis.

Insertion of the instrument: The tube should be well lubricated and inserted with a gentle rotary movement, the tip directed slightly back toward the sacrum and not forced in; this is especially the case with the hard tubes. Forcing the tube in might injure the mucosa. Do not press the tip of the tube against the wall of the bowel or direct the current against it, as either is irritating.

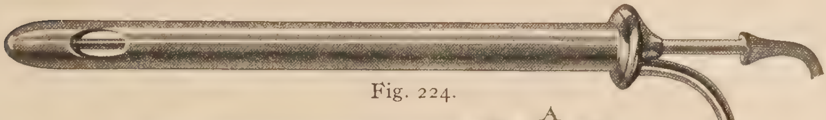

Fig. 224.

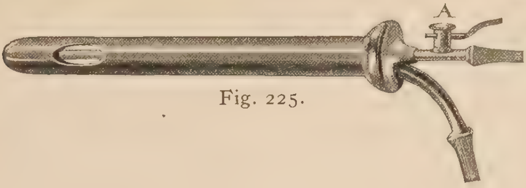

Fig. 225.

Fig. 224.—Kemp's rectal irrigator. All metal tube.

Fig. 225.—Electric attachment for electro-enteroclysis. Hard-rubber tube with metal center.

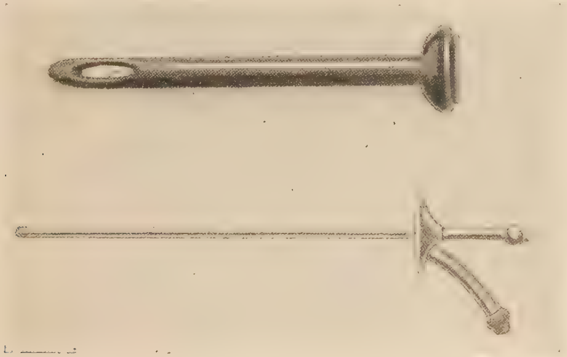

Fig. 226.—Kemp's tube (ready for cleansing). Hard rubber with metal center. Three sizes.

(3) If the flow ceases, rotate the tube slightly or withdraw it slightly while rotating and push it back. If the return tube seems plugged, attach the fountain syringe to it for an instant, and force the current in the opposite direction.

The best method to clear the outflow tube is as follows: A small hard-rubber or glass rectal syringe (enema) which contains from $\frac{1}{2}$ to

1 ounce can be employed. Shut off the entering current and then force water back into the bowel through the outflow tube. This will free it from the obstruction.

Length of insertion of the irritagor: The rectal tube should be inserted one-third to one-half its length in prostatic cases, etc., and full length for high irrigation.

If there is pain or difficulty in inserting the tube, a rectal examination should be made to see if there is any obstruction, such as an enlarged prostate, uterine fibroids, hemorrhoids, etc.

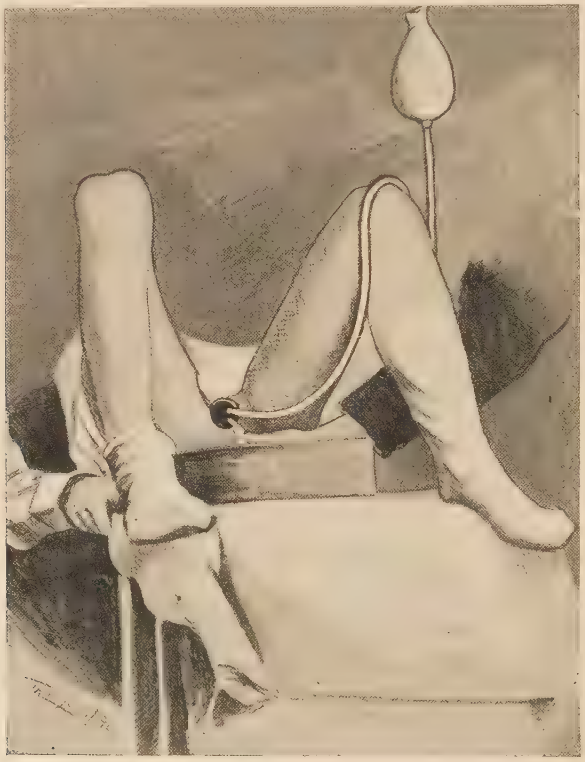

Fig. 227.—Enteroclysis (double current). Patient in dorsal position on bed-pan.

Withdrawal of the instrument: Do not withdraw the tube like an ordinary tip, as the mucosa might catch in the fenestræ. Withdraw it gently, and while doing so rotate it slightly first in one direction and then in the other. This prevents any such accident and frees the tube if it occurs.

Cleansing the irrigator: Unscrew the cap and withdraw the central tube.

*Position of the Patient.*—Elevation of the hips is the important feature, not the length of the tube. The different positions of the patients are illustrated and are readily understood (Figs. 227–231).

*Method by Rotation.*—Patient is placed on the left side with the hips elevated, and the descending colon is irrigated.

Rotate the patient gradually to the dorsal position, and then to the right side, with the hips elevated, the return tube being pinched. About 1½ pints to 1 quart (750–1000 c.c.) of fluid are allowed to run into the bowel.

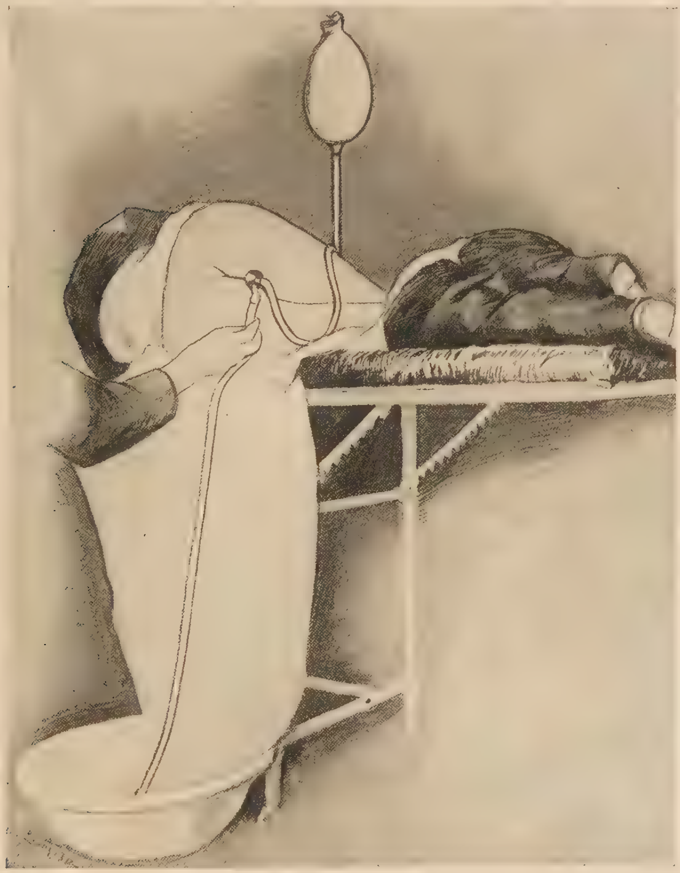

Fig. 228.—Enteroclysis (double current). Patient in Sims' position.

The shoulders are then elevated to above the level of the hips, the patient being still on the right side. This is to make the fluid gravitate into the caput coli.

The shoulders are then depressed to below the hip level, the patient on the right side; he is then gradually rotated to the dorsal position and then to the left side, and as a final step the shoulders are elevated, etc. In other words, the process is reversed. The return tube is then released and the fluid is allowed to escape.

Douglas H. Stewart of New York has devised an ingenious and convenient method. For continuous irrigation, he partly fills a wash-boiler with normal salt solution at the desired temperature, and this is kept hot by the addition of fresh hot saline solution; he hangs a gallon fountain syringe about 6 or 7 feet above the patient. He employs a hand-pump to force the solution from the wash-boiler through a connecting tube to the fountain syringe above. This simplifies the entire procedure and one does not have to disturb the bag in order to refill it. In addition, the outflow tube of the irrigator is left open, as there is sufficient force on account of the height of the bag to carry the fluid well up the intestines. Distention is carefully watched for and the entering current shut off in such event. The author has had this method employed on himself with excellent results.

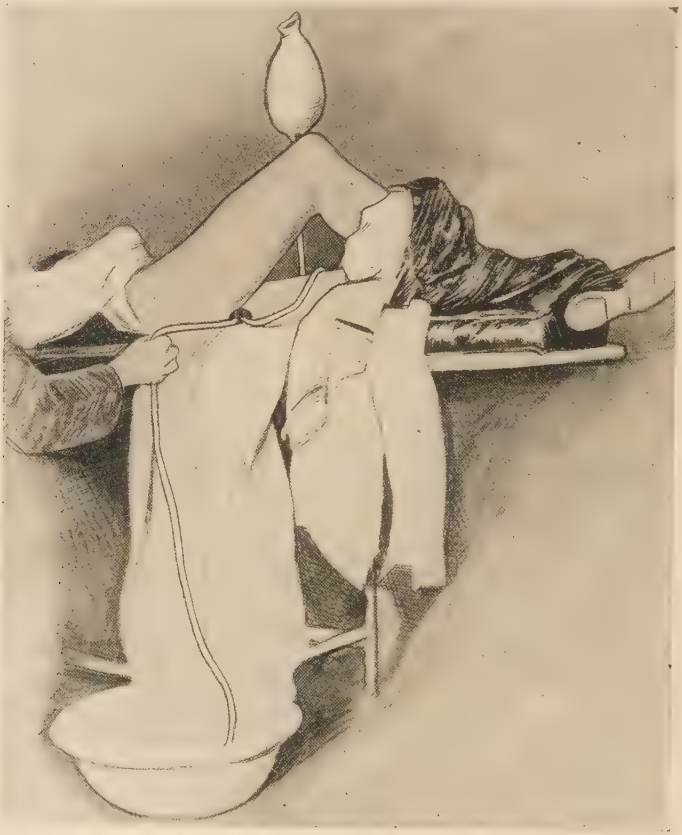

Fig. 229.—Enteroclysis (double current) without the bed-pan.

I employ *most frequently the dorsal position without the bed-pan.*

Temperature of the solution depends upon the conditions of its employment, an average of 101° to 105° F. in intestinal catarrh;

at 110° F. in typhoid for an additional eliminative effect, and for shock and uremia at 115° to 120° F. In the latter conditions continuous enteroclysis may be kept up for one-half to one hour, about ½ to 1 pint of hot saline solution being kept in the bowel continuously.

*Solutions Employed.*—Flaxseed tea (2 drams—8.0—of flaxseed to 1 quart—liter—of water, boiled twenty to thirty minutes and strained). Temperature, 101° to 103° F. This should be rather thin and oily in order to flow easily from a fountain syringe. Dilute it with boiled water if too thick. Normal saline solution with oil of peppermint, 5 to 15 minims (0.296–0.88 c.c.), or oil of cinnamon, 5 to 15 minims (0.296–0.88), to 1 pint (500 c.c.). Listerin, borolyptol, glycothymolin, borax, boric acid, bicarbonate of soda, tannic acid, tannin, and alum have been employed at a strength of 1 dram (4.0) to 1 quart (liter), and gomenol, 15 to 20 minims (0.88–0.00) to 1 quart (liter).

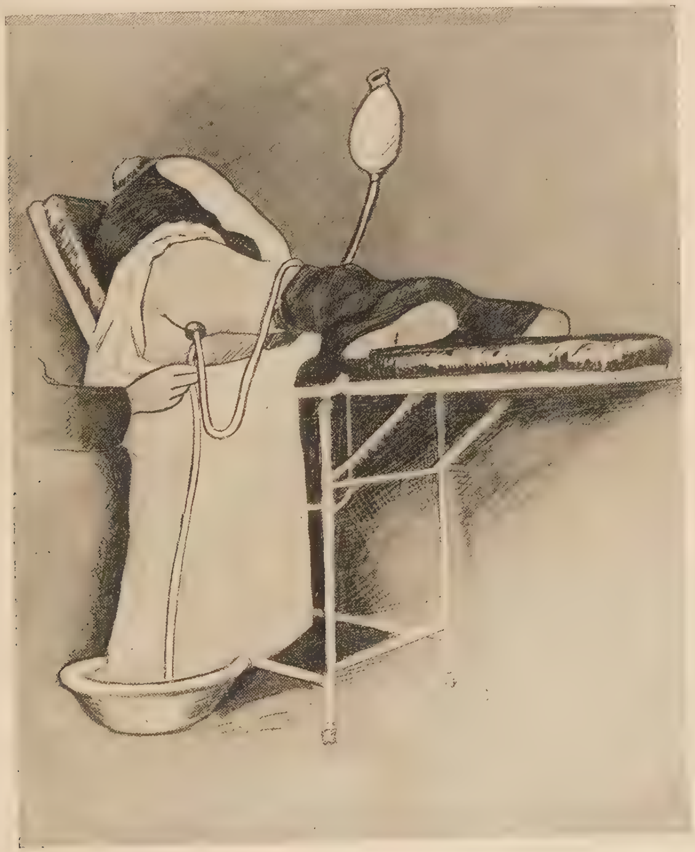

Fig. 230.—Enteroclysis (double current). Patient in semi-oblique position, as in pulmonary edema, etc., when dorsal position is impossible.

Solutions of nitrate of silver, 15 grains to ½ dram (1.0–2.0) to the quart (liter), have been used in chronic dysentery. Permanganate of potash, 2 to 5 grains to 1 quart of water, is useful when there is foul odor or much fermentation. Douglas H. Stewart recommends the following when there are foul odor, fermentation, putrefaction, and excessive bacteria:

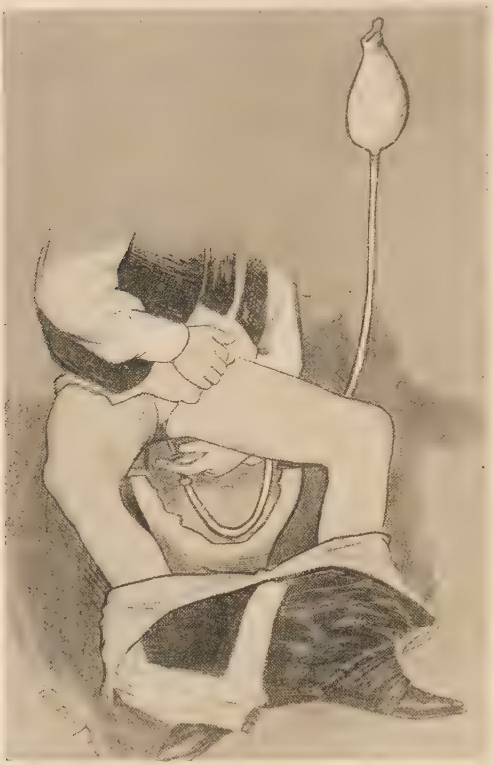

Fig. 231.—Enteroclysis (double current). Self-irrigation of the prostate per rectum.

| | |
|---|---|
| Chlorinated lime | 2 teaspoons; |
| Commercial acetic acid | 1 tablespoon. |

Dissolve in 1 quart of water.

Then add 9 quarts of plain sterile water. Salt is incompatible with acetic acid. Requisite portion of the 10 quarts is used for irrigation. The solution eliminates chlorin and yet does not seem to irritate. If salt solution is desirable, omit the acetic acid and substitute 2 teaspoonfuls of commercial sulphuric acid thus:

| | |
|---|---|
| Commercial sulphuric acid | ʒij (8.0); |
| Chlorinated lime | ʒj (4.0); |
| Normal salt solution | 10 quarts (liters). |

Delafield has used bichlorid of mercury (1:10,000), 2 quarts (liters), for septic membranous colitis complicating typhoid fever, with a recurrent tube, with success. Gum-arabic solution is often of service. Special solutions are described appropriately.

Normal saline solution, 1 dram (4.0) of salt to 1 pint (500 c.c.) of water, has a wide field of usefulness.

Enemata and enteroclysis are of value in dysentery, intestinal hemorrhage,[1] intestinal catarrh, typhoid, intestinal colic, tympanites, intestinal toxemias, intestinal dyspepsia, diarrhea, thirst, constipation, impaction, intestinal paresis, and jaundice.

They are extremely useful in shock, oliguria, uremia, sepsis, renal colic, and in inflammatory conditions of the genito-urinary organs of both sexes.

Irrigation may also be cautiously employed with cool saline solution at 90° to 50° F. to aid in the reduction of temperature. There is an element of danger in the prolonged use of very cold water for this purpose, shock or urinary suppression being possible in asthenic cases.

**Proctoclysis.**—In conclusion, I wish to refer to proctoclysis, the injection of normal saline solution into the rectum by the drop method, as first suggested by Dr. John B. Murphy of Chicago. This procedure is of special value in sepsis, and is of use as an adjunct to other treatment in postoperative shock, intestinal paresis, and uremia. In my experience the employment of continuous (recurrent) irrigation, with a temperature of the saline solution at 120° F., is more efficacious in the latter conditions, followed by proctoclysis as an adjunct.

One of the difficulties which the physician must endeavor to overcome in the administration of saline solution by the rectum or by infusion is the maintenance of a constant temperature of the solution. Elbrecht's apparatus necessitates a special heating chamber in addition to the containing reservoir, with the employment of an electric heater, an alcohol lamp, or a Bunsen burner. The method, though

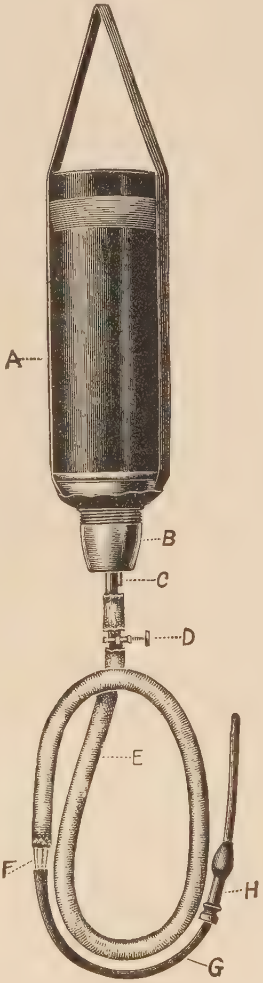

Fig. 232.—Heat-retaining proctoclysis bottle.

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[1] This refers to hemorrhage in the large intestine, where an extremely hot or cold astringent enema is of service.

scientific, seems complicated and is quite expensive. There are many other devices.

I have for some time employed the vacuum bottle[1] with a specially devised attachment, which has proved efficacious in preserving the saline solution at a constant temperature. It is readily understood from the illustration (Fig. 232).

Through the screw cap (*B*), which closes the bottle, passes a small hard-rubber conducting tube, to which is attached the outflow tube (*E*). Parallel with this is the filiform tube (*C*), which allows the entrance of a fine column of air, so as to render the flow possible. This last tube passes through the solution to within about $\frac{1}{8}$ inch from the bottom of the bottle. As the instrument is employed inverted, it would correspond to the same distance from the top of the bottle. This filiform tube is of hard rubber externally where exposed to the air as a non-conductor of heat. The part lying within the bottle is purposely made of metal, so that it is rapidly heated by the surrounding solution, and the entering air is thus, in turn, heated markedly.

A series of experiments have demonstrated that there is only a loss of 1° to 2° F. in the temperature of the solution in the bottle during the administration of proctoclysis (the drop method) lasting half an hour, a negligible amount. The screw compression valve (*D*) is applied close to the bottle attachment, so as to avoid as much as possible the solution cooling in the soft outflow tube. This outflow tube (*E*) is joined to the catheter (*G*) by a short piece of glass tubing (*F*), for the purpose of observing whether the flow is constant. The catheter for rectal injection passes through a self-retaining rectal tip (*H*), and the former can be inserted to any length desired. The conducting tube (*E*) is especially thick, as in Elbrecht's apparatus.

An asbestos tube surrounds the conducting tube from its junction at the bottle to the catheter. This lessens dissipation of heat and obviates the use of hot towels. The asbestos wrapping can be occasionally slipped off the glass connecting joint, so as to observe the flow. The vacuum bottle is filled in the usual manner and the special cap with attachment screwed on. The bottle is then inverted and suspended in a cord sling, as in the illustration. A small amount of fluid will escape from the bottle by the filiform air tube until the solution reaches the level of the tube, which now lies near the top of the bottle. The bottle is then suspended about 6 inches above the rectum or higher if desired, and the flow tested for the proper speed before inserting the rectal tip and catheter.

If flatus occur, lower the reservoir for a brief period to below the level of the abdomen, so the gas may escape into the bottle. At times, however, it may be necessary to remove the tube for a short period. The insertion of a T-tube between the reservoir tube and rectal catheter is of advantage. A short piece of rubber tubing is attached to the branch and immediately clamped. On occurrence of flatus, the lateral clamp is removed and the gas allowed to escape. It is then reclamped. This obviates removal of the tube. As already stated, there is practically no loss of heat in the container, all of it occurring during the passage of the drops through the outflow tube; the slower the speed, the greater the loss.

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[1] New York Med. Jour. and Philadelphia Med. Jour., Aug. 14, 1909.

At the start the speed is always more rapid, and, though gaged to, say, 15 drops per minute, may, in the course of two minutes, drop to 5. A test of two to three minutes should, therefore, be made before inserting the catheter, so as to insure a constant flow at the desired rate. The following table will be found of service; with

| Temperature of water in bottle. | Length of tube (inches). | Number of drops per minute. | Temperature in rectum. |
|---|---|---|---|
| 190° F | 30 | 60 | 115° F. |
| 160° F. | 30 | 20 or less | 100° F. |
| 150° F. | 30 | 40 to 50 | 100° F. |
| 138° to 140° F. | 30 | 150 to 200 | 105° to 110° F. |

If the injection is given at a greater speed than 200 drops per minute, the solution in the bottle should not be over 120° F., as there is practically no loss of temperature. This method by enema or recurrent enteroclysis would be of great value in shock. It could then be followed by proctoclysis as an adjunct.

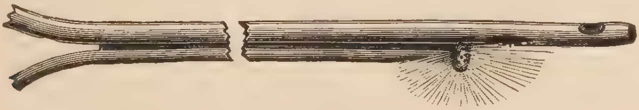

Fig. 233.—Türck's double-current needle douche for the sigmoid.

*Hypodermoclysis.*—There is a loss of 10° to 20° F. during the injection, depending upon the size of the hypodermic needle.

*Infusion.*—Dawbarn advocates a temperature of 115° to 120° F., preferably the latter; time, ten minutes to the liter (quart).

With the small vacuum bottle, containing about 1 quart (liter), a glass V tube can be inserted between the conducting tube and the rubber tube for attachment to the infusion canula. By this means it is possible to tell when the bottle is empty and thus prevent the entrance of air. A clamp can be applied close to the V tube on the canula side and the bottle refilled, the V tube being refilled before the conducting tube is reattached, and the latter being done while the solution is flowing.

A larger bottle can be secured for infusion, but the smaller one can be employed with these precautions.

The temperature of the saline solution does not practically change during the infusion, and should be at 115° to 120° F. in the reservoir.

**Needle Douche; Nebulizer; Colonic Massage Bags; Gyromele.**—These instruments[1] have been devised by F. B. Türck.

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[1] Jour. Amer. Med. Assoc., May, 1895.

The colon needle douche (Fig. 233) I believe of some value for local treatment by the alternate hot and cold spray in the atonic type of constipation.

The nebulizer (Fig. 234) is recommended by its inventor for spraying oils of cloves or cinnamon into the colon for their antiseptic, analgesic, and vasomotor effect.

He recommends distensible bags for massage of the atonic sigmoid and rectum, which I do not advocate.

The gyromele, already described, Türck recommends in the rectum and sigmoid for cleansing the mucosa and producing vibratory movements. I question the possibility of its entering into the sigmoid flexure except on rare occasions.

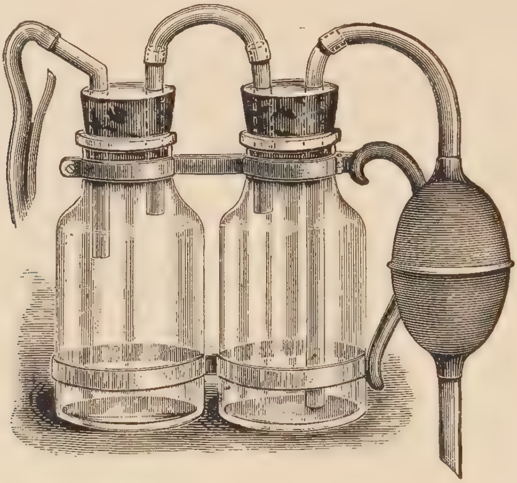

Fig. 234.—Türck's nebulizer.

**Massage, Gymnastics, and Exercise.**—The general methods of massage have been described. The course of the colon should be followed. The cannon-ball and vibratory massage are of service. The portable Vedee vibrator is a useful instrument for the general practitioner. It is illustrated in Part II of this volume. Gymnastic exercises and out of-door sports are valuable for strengthening the abdominal and intestinal musculature, especially in intestinal atony with constipation. Rowing with a sliding seat, golf, horseback-riding, bicycling, and walking are useful.

*Mechanical Support.*—Adhesive strapping (Rose's belt), abdominal supporters, etc., are indicated in enteroptosis, in atonic conditions of the intestines or of the musculature of the abdominal wall, in hernial protrusions, etc.

**Hydrotherapy.**—The Priessnitz compress, poultices, and the application of heat or cold locally are useful for pain.

Sitz-baths and abdominal douches are employed as already described.

**Electricity.**—Galvanic, faradic, and static electricity may be employed percutaneously. The faradic, chiefly in atonic conditions; the galvanic, in painful neurotic affections. Static electricity can be used for both purposes.

About ten to fifteen minutes is the average period of treatment. When externally applied the current should follow the course of the colon and then be given over the small intestine. Electric vibration may be used.

Violet rays, the high-frequency current, etc., have been advocated for various conditions, but further investigation, I believe, is necessary before they can be recommended. The application of heat and light (electric-light bath) to the abdomen in painful conditions due to a gouty or rheumatic tendency, or in enteroptosis or mucous colic its application to the sensitive areas may prove of service.

*Intrarectal Electricity.*—Boudet's electrode presents the disadvantage that sometimes frequent evacuations necessitate withdrawal of the electrode.

The author's instrument (Fig. 225) consists of a recurrent irrigator, external tube hard rubber, internal tube metal, with an attachment for a battery pole; a sponge attached to the other pole is placed over the abdomen. Continuous enteroclysis is given with hot normal saline solution, the water carrying the current.

The treatment should be given for five to thirty minutes, depending on the indications. It is excellent for simple atonic constipation and for intestinal paresis. The faradic current is preferable, as strong as the patient can bear. The galvanic may be employed, with negative pole in the rectum, with a current of 10 to 15 milliampères.

## CHAPTER XXI

# DIET; INTESTINAL DYSPEPSIA; INDICANURIA; SACCHAROBUTYRIC PUTREFACTION; BOTULISM; HYDROGEN SULPHID AUTO-INTOXICATION; ENTEROGENIC CYANOSIS; METEORISM; ENTERALGIA; VISCERAL ARTERIOSCLEROSIS; SENILE DYSPEPSIA; ANOMALIES; INTESTINAL SAND

### DIET

The general principles of diet were described in Diseases of the Stomach. I will briefly mention a few general rules which should be applied.

*Acute intestinal disorders must be managed* on the principle of rest. Light food (chiefly liquid) should be given, such as broths, barley-water, gruels, koumiss, matzoon, bacillac, lactone-buttermilk, and in some cases milk, but diluted with barley-water or lime-water. It has been demonstrated that in the acute intestinal catarrh of infants and children, undiluted (or at times even diluted) milk will frequently pass undigested and intensify the inflammatory process, so that reliance is placed for a time on barley-water and similar preparations.

Seibert has demonstrated conclusively that milk is not the ideal food for typhoid fever.

In chronic intestinal disease, for a brief period, rest may be given to the intestines by means of a fluid diet, but feeding should soon be increased, the general nutrition improved, and the intestinal tract gradually accustomed to a regular diet, if such be possible. Milk, cream, raw eggs, carbohydrates, and fats are of value to improve nutrition.

In these chronic cases especially, and also in intestinal dyspepsia, all or some special digestive function may be disturbed, such as for proteins, fats, or carbohydrates. The presence of indicanuria, the test-diet with stool examination, the fermentation test, together with the clinical symptoms, will determine the type of disturbance of the intestinal digestive function. *The findings would modify the diet.*

*Dietetic measures* are of special value in reference to disturbances accompanied by constipation or diarrhea.

1. *Laxative foods* are articles which increase intestinal peristalsis. Among such are fruit juices and raw and cooked fruits, such as apples, pears, plums, peaches, strawberries, gooseberries, dates, and figs. Salads, garden vegetables, due to water contained and indigestible

residue, such as melons, cucumbers, spinach, tomatoes, pumpkins, and cabbage; cider, bonny-clabber, koumiss, matzoon, and bacillac, through formation of acid products and gas, increase peristalsis. Some of the fruits and vegetables mentioned have a similar effect. Brown bread and oatmeal tend to move the bowels, also water or carbonated waters. Sugar has a laxative effect. Considerable fat, in the form of cream, butter, or olive oil, aids bowel action.

2. *Constipating Foods.*—Among such are substances containing astringent agents, especially tannic acid, such as tea, dried bilberries, French red wines, cocoa, the acorn preparations, such as acorn coffee, acorn cocoa; mucilaginous foods, such as sago, tapioca, rice, and barley; also foods which leave little residue and exert no irritation, such as albumin-water (white of egg dissolved in water), scraped raw beef, mutton broth, etc.

*Pathologically, excess of carbohydrates* may be a cause of diarrhea, as may *excess of proteins.*

Milk is constipating in one person, laxative in another, and has no special effect on others. Boiled milk is usually constipating.

Dietetics have changed markedly during the last few years. In chronic colitis with diarrhea the chief diet was formerly scraped meat. Modern methods allow a much greater variety, and the employment of mashed potatoes and boiled rice lessen peristalsis and are often of considerable value.

*Certain foods*, when taken under ordinary conditions, have no marked influence in increasing peristalsis; among these are meats, fish, meat powders, artificial foods, such as peptone, nutrose, somatose, sanatogen, plain or flavored; eggs, well-baked bread (wheat), crackers, zwieback, and butter or fat in small amount.

*Marked seasoning* of foods increases peristalsis. The finer the particles of food, the less the irritation; the coarser they are, the greater stimulation they produce on the intestines.

*Gelatin.*—I have referred to the value of gelatin in ulcer of the stomach and in hyperchlorhydria. Gelatin does not build up new tissue, no matter how much is ingested, though it may diminish tissue waste (Voit).[1] It cannot be reconverted into a protein.

Kirshmann[2] shows that gelatin *spares protein in metabolism.*

The ingestion of 7.5 per cent. of the total heat requirement of the organism in the form of gelatin spares 23 per cent. of the body's protein, while 60 per cent. gelatin reduces it 35 per cent. One gram of gelatin furnishes 4.1 calories. About 50 grams of gelatin represents this requirement in a person weighing 154 pounds. The small amount of gelatin has nearly as much effect as larger quantities. Its value in typhoid is, therefore, evident to lessen *nitrogen excretion.*

Kaufmann[3] shows that when the lacking tyrosin, cystein, and tryptophan are mixed with gelatin in the proportion in which they occur in true protein, and are given to a dog or man, nitrogen equilibrium may be established.

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[1] Hermann's Handbuch, Stoffwechsel, 1881, p. 396.

[2] Zeitschrift für Biologie, 1900, Bd. xl, p. 54.

[3] Pflüger's Archiv., 1905, Bd. clx. p. 440.

Gelatin does not yield indol, and can be employed in the form of jellies as a nitrogenous substance to replace proteins in cases of indicanuria (Herter).[1]

In severe inflammation of the intestines artificial feeding may be necessary, by rectum or subcutaneously.

Rectal alimentation has been described under Diet, in Part II.

Sterile olive oil, 1 to 2 ounces (30.0–60.0), may be injected subcutaneously two or three times a day between the crest of the ileum and lower border of the ribs (preferably). Sterile almond oil in doses of 1 to 2 drams (4.0–8.0) may be substituted. I do not advocate, however, the subcutaneous method.

Normal saline solution, 1 pint (500 c.c.) to 1 quart (liter), may be slowly injected in the same region in case of collapse or hemorrhage.

## INTESTINAL DYSPEPSIA

With intestinal catarrh or biliary or pancreatic obstruction there is perversion of intestinal digestion. These conditions will not be considered here.

Digestive processes in the intestine may become abnormal when the intestinal contents are no longer normal. This may occur even with *intact mucosa and with sufficient bile and pancreatic juice.* Indigestible food, or excess in some special types of food, may lead to fermentative or putrefactive processes in which bacteria take part, and may produce dyspeptic symptoms.

Indicanuria or saccharobutyric putrefaction occur from these conditions.

There may be functional disturbances of pancreatic digestion for the proteins, carbohydrates, and fats, or any one or two of these functions may be disturbed.

Riegel describes cases with marked fermentation of the carbohydrates. There is a jejunal diarrhea of gelatinous semifluid character, often quite green, with bile-pigment reaction and acidity. There is mucus in these movements, but no formed elements, such as epithelial and round cells, which occur in catarrhal conditions. Catarrh may ultimately result.

Herter[2] shows that there is an intestinal indigestion in children of marantic type, in which there is intolerance to carbohydrates, and light-colored and gray or fatty stools occur. Indican and phenol are found in large amount in the urine. A great number of Gram-positive bacilli of the Bacillus bifidus type occur in these cases.

Schmidt and Strassburger describe "intestinal fermentation" as dyspepsia in which the stools are light yellow, foamy, with the odor of butyric acid. Fatigue, discomfort, loose stools, or even diarrhea may occur. Abdominal pains, distention, and some tenderness may be present. These cases correspond to saccharobutyric putrefaction (Herter).[1] Dyspeptic symptoms may be due to the decomposition of proteins.[2] Various symptoms, as stated, may be present with intestinal dyspepsia, such as distention, pain, borborygmi, passage of flatus, feeling of discomfort in the abdomen. Diarrhea or irregularity of the bowels is present; loss of appetite, eructations, nausea, and even vomiting may occur. The best method of determining the intestinal functions is by the Schmidt–Strassburger *test-diet*, with *examination of the stool.*

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[1] Bacterial Infections of the Digestive Tract, p. 267.

[2] Ibid., p. 285.

**Treatment.**—That variety of food must be limited for which the particular disturbance exists. Liquid or semiliquid diet may be necessary. *Indicanuria* and *saccharobutyric putrefaction* must be *appropriately treated.*

Lactic acid has been recommended in the form of fermented milks, such as koumiss, matzoon, bacillac, kefir, or lactone-buttermilk; chicken, jellies, gruels, etc., can be given later.

Calomel, $\frac{1}{40}$ grain (0.0016) four times a day, is suggested by Van Valzah[3] for fermentation; or resorcin, 5 grains (0.3) t. i. d., is excellent.

Taka-diastase, 5 grains (0.3) t. i. d., is valuable in amylaceous dyspepsia; or pancreon, 5 grains (0.3) t. i. d., in disturbances of fat and protein digestion,[4] and pancreatin, same dosage. Cellasin, 5 to 10 grains (0.3–0.6), given t. i. d. an hour after meals, in combination with bicarbonate of soda, 10 grains (0.6), has of late proved valuable in amylaceous and fatty dyspepsia.

Holadin, an extract of the entire pancreas gland, represents all the constituents of the gland, both of the digestive and internal secretions. This may be given in a 3-grain (0.3) capsule. One would begin with one capsule immediately before or two hours after each meal, and increase to two capsules t. i. d. The writer usually adds a tablet of secretin or, preferably, prosecretin, 1 grain t. i. d., to the above to stimulate pancreatic secretion. As the bile salts intensify the action of the pancreas, one may at times substitute capsules containing holadin and bile salts (holadin, $2\frac{1}{2}$ grains; bile salts, $\frac{1}{2}$ grain in each capsule). One of these is given t. i. d. about three hours after meals. Enterokinase in 3-grain (0.2) doses may also be given. This product (Fairchild's) is still in the experimental stage. Enterokinase, a ferment of the duodenum, activates the trypsinogen. The writer usually first tries holadin with prosecretin and, lately, the addition of enterokinase.

In conclusion, I would advise that in the milder cases the special food stuff which causes dyspepsia should be limited in quantity, but not entirely cut off. Eggs, soft boiled or raw, vegetables in mashed forms, bread or crackers, stale and well broken up, and sanatogen are of service; butter or cream according to indication. Later the diet is increased.

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[1] Bacterial Infections of the Digestive Tract, pp. 294, 300.

[2] Ibid., pp. 280, 306.

[3] Med. News, Jan. 17, 1903.

[4] True Intestinal Dyspepsia (Einhorn), Amer. Jour. of Med. Sciences, November, 1907; also Med. Record, September 4, 1909.

## INDICANURIA

Indicanuria designates the presence of indican in the urine, as demonstrated by reactions with the formation of indigo, on using Jaffe's, Obermeyer's, or similar tests.

Indol is absorbed from the intestines and forms in the liver indoxyl potassium sulphate or indican, an ethereal sulphate which is eliminated in the urine.

There are various causes of indicanuria: excessive protein diet, catarrh of the small intestine, causing alterations in the mucosa and increased intestinal putrefaction, typhoid, cholera, etc., constipation, alimentary putrefaction, partial or complete obstruction of the common bile-duct, decrease of normal digestive fluids, intestinal obstruction, and peritonitis. Certain drugs, such as salol, salophen, and creosote, give reactions which must not be mistaken for it, while urotropin causes its disappearance.

Intestinal indicanuria is an evidence of intestinal putrefaction. Excessive quantity of protein, especially of meat, may be a cause. Any condition favoring stagnation in the intestines helps produce this condition. Imperfect action of a cathartic can produce indicanuria.

In children little indican appears. Many adults show indican and suffer from no symptoms, but this is true of constipation. Finally, persons with indicanuria show clinical evidences of intestinal disorder and sometimes symptoms of auto-intoxication, frequently affecting the nervous system. Neurasthenic and even melancholic symptoms may be dependent on this form of auto-intoxication. Headache, migraine, myasthenia, epileptiform seizures, early fatigue, cyclic vomiting, or progressive muscular atrophy may also be dependent on this condition (Herter).

Intestinal putrefaction may directly affect the liver and kidneys. I have a patient with marked indicanuria with *bile in the urine*, albumin casts, cylindroids, and diminished urea. Treatment of the intestinal putrefaction cleared up this condition.

This corroborates the view of W. H. Porter[1] that excessive intestinal putrefaction may cause disturbance of the hepatic cells. There was no jaundice in this patient. From personal experience, therefore, the author finds that marked indicanuria (intestinal putrefaction) may be directly productive of an acute nephritis, or produce an acute exacerbation in a case of chronic nephritis. This has been the experience of Wm. H. Thomson in a number of cases which he has reported. Under such conditions, I would advocate adherence to the most rigid form of diet for the indicanuria, chiefly the sour milks.

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[1] The Post-graduate, Oct., 1902.

After a practical disappearance of the indicanuria, with clearing of the acute condition, one should regulate the protein diet in the cases of chronic nephritis according to the amount of nitrogen excreted in the urine. Give the patient what he can take care of and no more. The colon bacilli, through their activity in the decomposition of proteins, are chiefly responsible for the production of indol. The Bacillus aërogenes capsulatus and the Bacillus putrificus, when present, favor the putrefactive action of the colon bacilli on the proteins.[1] Indicanuria, I believe, further influences the production of arteriosclerosis. I will briefly recount an interesting case. The writer was called in consultation to a patient suffering from persistent vomiting which apparently could not be relieved. On physical examination, evidences of arteriosclerosis and excessively high pulse tension were found. Blood-pressure was taken at once and found to be 250. Under the hypodermic administration of nitroglycerin, $\frac{1}{50}$ grain every two hours, the vomiting rapidly ceased and the pressure fell. Sodium nitrite was subsequently added to the treatment. The urinary analysis at this time showed a very slight chronic nephritis. The most interesting feature of the case was that the patient had during the course of the preceding two years several such attacks. Numerous examinations of the urine had been made by competent pathologists, and, excepting the excessive indicanuria, no evidences of nephritis had been found; but the high tension and arteriosclerosis were always present. Numerous eminent consultants had examined the patient, and all agreed that the indicanuria had an influence on the arteriosclerosis. The author believes the slight nephritis found, when he saw the case, to be undoubtedly of recent development, and that the indicanuria had a direct bearing on the production of the arteriosclerosis. There were no evidences of syphilis in this patient and the Wassermann test was negative. The treatment of this condition would be the treatment of indicanuria plus the reduction of blood-pressure by the use of the nitrites and tincture of aconite (old Pharmacopœia), 5 minims t. i. d., as recommended by Wm. H. Thomson, with the subsequent addition of the iodid of soda or of potash. Metchnikoff believes that his experiments have now established beyond question that small doses of paracresol and indol, acting on the organism over a longer or shorter period, are capable of inducing chronic lesions of the nature of sclerosis. Such lesions are the ones that are most frequently encountered in senility. He has demonstrated, he states, that the phenols and indol found in the urine and stool are not the excreta of our tissues, but are the products of the permanent microbian flora. It is not unreasonable to suppose that the digestive tract can constantly harbor an injurious flora, the source of chronic poisoning leading to arteriosclerosis. The colon bacillus and Bacillus perfringens produce poisons which are absorbed by the intestinal walls and are capable of inducing serious lesions in arteries, kidneys, liver, and other organs. His observations are described in the Annales de l'Institut Pasteur, Paris, October, 1910, xxiv, No. 10, pp. 753–831.

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[1] Herter, Bacterial Infections of the Digestive Tract.

Taylor[1] recently reports transient heart-block due to indicanuria.

The following test is reliable: Place in a test-tube equal quantities (10 c.c. of each) of urine and chemically pure concentrated hydrochloric acid. To this mixture add 3 drops of ½ per cent. solution of potassium permanganate. Then add a small portion of chloroform, 1 or 2 more drops of the permanganate solution, and a few drops more of chloroform, or a total of 75 drops (5 c.c.) of chloroform, and shake vigorously for a few seconds.

Indigo-blue is deposited in presence of this indicator. Bile should be tested for, even if there are no evidences of jaundice. W. H. Porter's scale (Plate I) is a basis of comparison in the absence of bile test.

Rosenbach's test, which consists in boiling the urine with nitric acid, gives a Burgundy red if putrefaction is present. This may occur when no indican is found. It is due to substances of a like class.

**Treatment.**—*Diet.*—Matzoon, koumiss, lactone-buttermilk, bacillac; later, stale crackers with butter, boiled rice, and jellies (gelatin) are to be added. Herter shows that clinically the carbohydrates have an influence, and the substitution of a quickly digested carbohydrate, like rice which has been well cooked and forced through a colander, or large quantities of bread or sugar, will lessen the excretion of indican. Taka-diastase or cellasin can also be given to aid their digestion.

*Medication.*—Blue mass, 5 grains (0.3); calomel, 5 grains (0.3), once a week, and a saline cathartic for a short period daily.

Urotropin, 5 grains (0.3), with benzoate of soda, 10 grains (0.6), t. i. d., has cleared up many cases, albumin casts, bile, and indican disappearing within a short time. This shows the improvement was not due to the mere interference with indican reaction. Lactic acid bacilli tablets are useful. Fairchild's and those advocated by Metchnikoff have given me good results.

Aspirin, salicylate of soda, and salol, 5 to 10 grains (0.3–0.6), are of service, as is also chologestin, 2 to 3 drams (8.0–12.0) t. i. d. The following has sometimes proved of value:

℞. Sulphocarbolate of soda ................ ʒiv;
Permanganate potass.................. gr. xlviij;
Beta-napthol ........................ gr. xlviij.—M.
Div. in capsules No. xlviii. Shellac cover.
Sig.—One three times a day, after meals and at bedtime.

Enteroclysis is valuable, especially the injection of 1 pint to 1 quart (500–1000 c.c.) of a 1:1000 acetozone solution, with the patient in the knee-chest posture. This should be retained for a short time.

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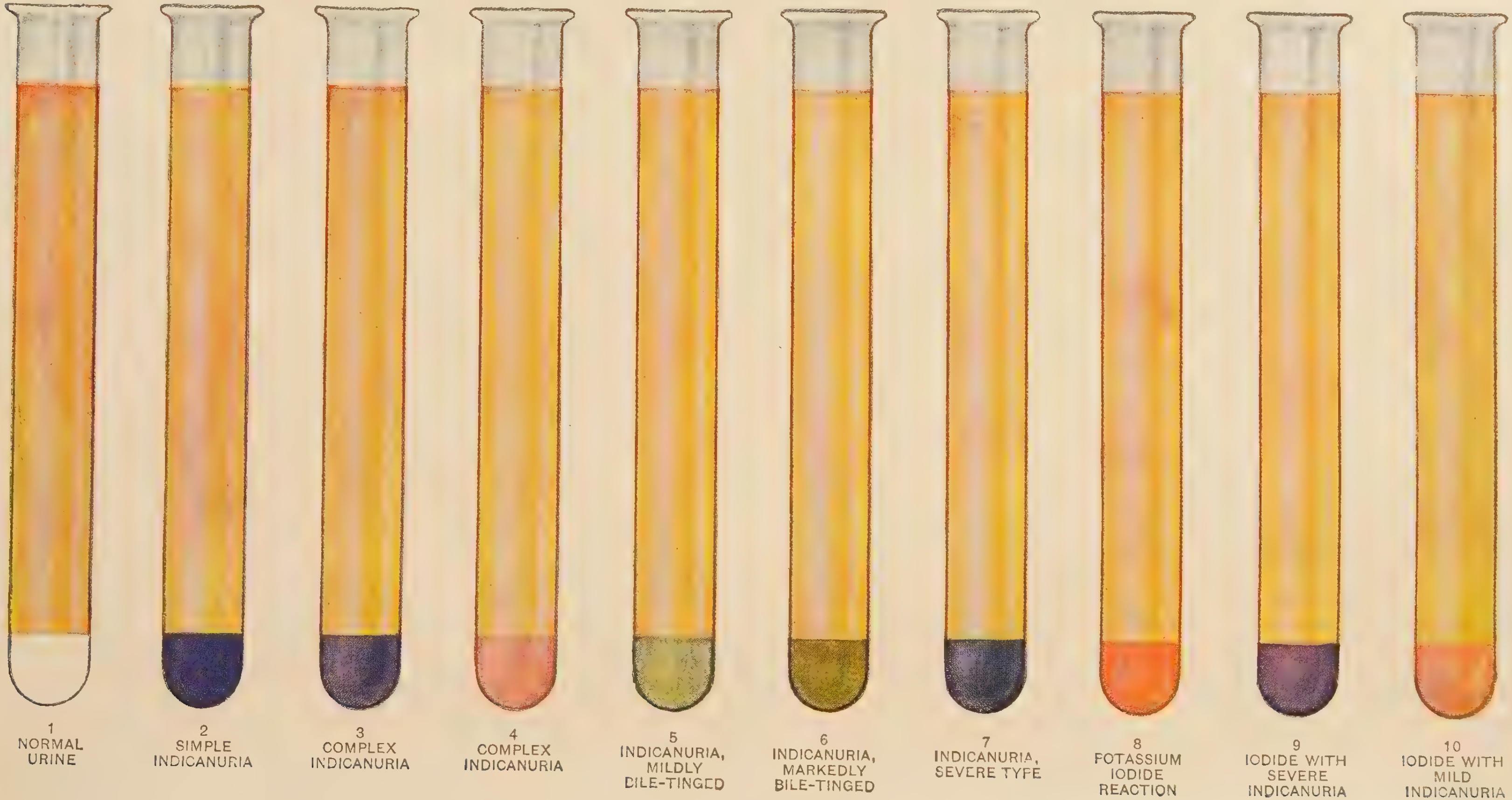
[1] Jour. Amer. Med. Assoc., April 18, 1908.

# PLATE I



INDICAN COLOR SCALE.

(Courtesy of W. H. Porter, M. D.)

This procedure can be carried out every day or few days. *Gastric disturbances should be investigated and corrected.*

George A. Tuttle has shown that potassium iodid, especially in cases with arteriosclerosis, will lessen indican. Basham's tincture has been of benefit in some. Some have advocated acetozone solution (1 : 1000), 1 pint (500 c.c.), in divided doses daily by mouth.

Bassler[1] recommends rectal instillations of autogenous bacteria and strains of the colon bacillus in chronic intestinal putrefaction, but the writer does not advise the method. The author *recommends urotropin treatment combined with lactic acid bacilli tablets and sour milks.*

## SACCHAROBUTYRIC TYPE OF INTESTINAL PUTREFACTION

This type of decomposition is produced chiefly by the Bacillus aërogenes capsulatus,[2] Gram-positive. Bacillus putrificus or Gram-positive diplococci may at times be associated.

The Bacillus aërogenes under anaërobic conditions can attack carbohydrates and proteins. Butyric acid is often formed in considerable amount, carbon dioxid, hydrogen, ammonia, and often propionic, caproic, or valeric acids. The odor of the movements is often intense and characteristic of butyric or caproic acid. Excessive gas is liberated. The seat of this process is chiefly in the large intestine and lower ileum.

When proteins are attacked there is less gas liberated. With excessive gas production the feces have a low specific gravity and float on water. There are small bubbles in the contents and the feces are a light color. Patients who suffer from this condition do not tolerate well either carbohydrates or acids, flatulence and diarrhea occurring after use of cereals, starchy food, and especially sugar. The mucous membranes of the digestive tract are easily irritated, and there may be epithelial desquamation in the mouth. Howard[3] has described instances of superficial necrosis of the mucous membrane of the stomach and intestines, associated with an abundant presence of the Bacillus aërogenes capsulatus. These necrotic areas most often lie beneath the folds of the valvulæ conniventes and may occur with gas cysts. It does not seem likely that these bacilli are responsible for severe acute inflammatory lesions of the intestines, but more probably for the subacute enteritis that is so often present in cases that show large numbers of the bacilli in the stools. There are, moreover, many instances of acute diarrhea associated with free Bacillus aërogenes capsulatus multiplication and with severe primary anemia, and in children capsulatus infection may lead to the development of extreme anemia with general edema. The Bacillus aërogenes capsulatus is an active hemolysing agent[1] and many instances of "primary" pernicious anemia and of secondary anemia show pronounced indications of excessive saccharobutyric putrefaction (infection with the Bacillus aërogenes capsulatus), and probably this infection has a causal relation to the anemia. Streptococci (sometimes of oral origin) are at times found in some of these anemias in addition to the Bacillus aërogenes capsulatus, and the anemia may be due to the combination. In the simple cases indol is generally absent.

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[1] Med. Record, Sept. 24, 1910.

[2] Herter, Bacterial Infections of the Digestive Tract.

[3] Contributions to the Science of Medicine, dedicated by his pupils to Wm. Henry Welch on the 25th anniversary of his doctorate, Baltimore, 1900, p. 461.

*Combined Saccharobutyric and Indolic Type.*—The Bacillus aërogenes capsulatus is also able to break down proteins into a suitable form for the use of other putrefactive bacteria, among which are the indol-forming organisms.

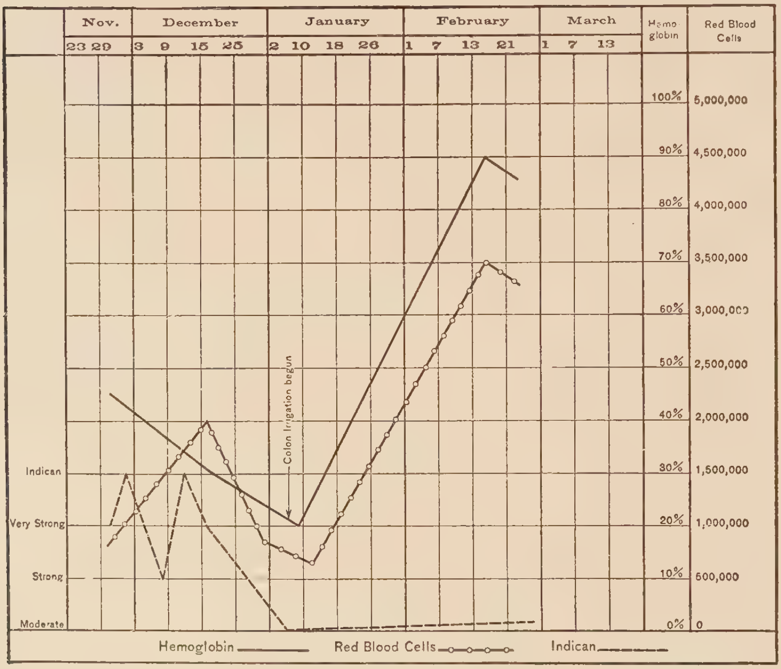

Fig. 235.—Chart of pernicious anemia demonstrating value of enteroclysis.

This last form, associated with the excessive production of indol, is the most severe type, and these cases are subject to auto-intoxication, depressive mental conditions, and diminution in muscular power (muscle fatigue), according to Herter.

In the severe types the anaërobic organisms, especially the

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[1] On Bacterial Processes in the Intestinal Tract in Some Cases of Advanced Anemia, etc. (Herter), Jour. of Biological Chemistry, vol. II, Nos. 1 and 2, Aug., 1906.

Bacillus aërogenes capsulatus, produce hemolytic substances which are believed to have a *bearing on the production of pernicious anemia.*

The results of intestinal irrigation in such cases have been favorably reported by Hollis[1] and Ditman,[2] as in Fig. 235, with marked improvement in the hemoglobin and red cells.

Arthritis deformans[3] has recently been imputed to putrefactive changes in the intestinal canal. At the Red Cross Hospital excellent results were secured by the author by enteroclysis and fermented milks, no medication; the swelling in the joints rapidly subsiding.

**Treatment.**—Careful mastication of the food, cleanliness of the mouth, and correction of gastric disturbances are necessary. Sugars should be omitted, starchy food cut down, and meat diminished or eliminated if the mixed type with indican is present.

Taka-diastase, 5 grains (0.03) t. i. d., aids starch digestion. Pancreatin preparations may be tried. Holadin, 3 grains (0.2), with presecretin, 1 grain, and enterokinase, 3 grains (0.2) t. i. d., may be substituted. Pancreon, 5 grains (0.3) t. i. d., is useful. The fermented milks are of special value. Lactic acid bacilli tablets are useful.

Enteroclysis, or high enemata, with saline solution, acetozone (1 : 1000) or hydrogen peroxid (1 : 1000), are of service. The latter are of special value.

Ichthoform, formidin, ichthalbin, aspirin, salol, salicylate of soda, 5 grains (0.33) t. i. d., are useful, or urotropin, 5 grains (0.3), with benzoate of soda, 10 grains (0.6) t. i. d. This last combination, with the addition of the high enema of acetozone, 1 quart (liter), strength 1 : 1000, I have found of great value. Permanganate of potash, up to 1 grain (0.06) t. i. d., may be of service. Herter suggests the possible value of dioxid of manganese. Surgery may be indicated in extreme cases. If dilatation of the stomach, not responding to medical treatment, is the cause, then gastro-enterostomy is indicated. With failure of medical treatment and progressive anemia and mental disturbances, Herter suggests appendicostomy followed by enteroclysis. I am opposed to shortening or short circuiting the large intestine for this condition. In extreme cases Gant's cecostomy, with irrigation of colon and ileum, might be of service.

## ACIDOSIS (ACETONURIA)

Since acidosis involves the *problems of metabolism* and is considered a toxemia, a brief reference to the subject is included for convenience under Intestinal Auto-intoxication. The healthy performance of the functions of the tissues depends on certain chemical processes taking place in solutions of a certain composition, the chemical reaction of which is faintly alkaline. Any disturbance in metabolism which tends to reverse the condition, that is, to *diminish* the alkalinity of the juices, may set up a *condition of acidosis*. This *disturbance does not* necessarily lead to an actual reduction of the alkalinity of these fluids; the blood, for example, since considerable quantities of acid may be produced in the body by abnormal metabolic processes, and yet lead to no appreciable alteration in the reaction of the tissues or the blood, being neutralized by the alkalis in the body fluids and cells and by ammonia produced from the hydrolysis of proteins, which would, in the absence of the call for an alkali, have been converted into urea and excreted in that form. If the amount of acid ingested or produced in the body is so great that it cannot be neutralized or disposed of by oxidation, then the reaction of the blood can no longer remain unaltered, there will be a reduction of its alkalinity, and there is danger of the condition passing from the milder one of acidosis to one of acid *intoxication*.

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[1] Herter, Bacterial Infections of the Digestive Tract.

[2] Med. Record, Feb. 2, 1907.

[3] A Preliminary Report on the Relation of Albuminous Putrefaction in the Intestines to Arthritis Deformans, by Andrews and Hoke, American Journal of Orthopedic Surgery, July, 1907; also Cornwall, Med. Record, April 1, 1911.

Acidosis is characterized by excretion in the urine of an excess of acid radicles, acetone alone in the milder cases, and diacetic acid in addition in the more severe types, and these two, with B-oxybutyric acid as well or B-oxybutyric, as the chief in the most severe cases. There is sometimes the characteristic sweet odor on the breath from the acetone, particularly in diabetes or with acid intoxication.

The simplest test for determination of acidosis is the discovery of one or more of these acid radicles—acetone alone or the others in addition—in the urine. The amount of ammonia in the urine is also a valuable indication of the degree of acid poisoning, and the odor of acetone on the breath.

The reduction of the alkalinity in the blood further shows there is danger of acidosis passing into acid intoxication. In severe cases of acidosis the reaction of the urine to an alkali serves as a test as to the comparative severity of the process. A large amount of bicarbonate of soda may be taken without producing neutrality or alkalinity of the urine in severe cases.

As acidosis may exist for a long time without *any symptoms*, in some cases for a considerable period, and in others with none, except that the patient may be *subject to digestive disturbances*, and since this mild type of acidosis may at any time become severe and even result in dangerous acid intoxication, the *urine should always be examined for acetone as a matter of routine*. This is particularly true in early pregnancy and in all patients about to be operated upon. The presence of even a small amount of acetone previous to operation would contraindicate chloroform, and also indicate preparation of the patient by the administration of soda bicarbonate previous to operation. Other anesthetics may produce acetonuria as well as chloroform. In fact, the writer believes the administration of an alkali (soda bicarbonate) several days before operation is an excellent procedure.

James Ewing[1] divides cases of acidosis into two main groups: one *without fatty degeneration of the liver*, as represented by diabetic acidosis, and one with fatty degeneration, represented by the toxemia of pregnancy, delayed chloroform-poisoning, Eck's fistula, cyclic vomiting in children, and poisoning by hematoxic immune serum.

Acetone bodies are found in the urine in a large number of other clinical conditions in gastro-intestinal diseases, diseases of the liver, gastro-intestinal diseases accompanied by inanition, starvation, in some cases acute yellow atrophy, phosphorous-poisoning, etc. Park[2] reports cases of ileocolitis with acidosis and marked respiratory disturbances, and Singer[3] a case of severe auto-intoxication with acetonuria, extreme bradycardia, and convulsions.

Deprivation of the carbohydrates and dependence on protein and fats results in the development of acetone bodies from the latter.

The ingestion of carbohydrates is followed by lessened acetonuria, and advantage has even been taken of this in the acidosis of diabetes by giving temporarily an oatmeal diet to relieve the acetonuria and lessen the chance of a subsequent diabetic coma. Disturbance of the liver functions, with interference with metabolism as a result, have, therefore, an influence in many cases as noted by Ewing. In view of the occurrence of acetonuria with intestinal auto-intoxication and ileocolitis, etc., and the probable damage to the liver cells by the toxins, further interference with metabolism with acetonuria resulting, would readily suggest itself. Postoperative vomiting from acidosis may be a decided menace to the patient. The writer has recently treated two severe cases of Virgil P. Gibney, following the *administration of ether*. It occurs most frequently after chloroform, but also after ethyl chlorid and nitrous oxid.

These cases were both girls, aged eight and nine years, with a previous history of weak stomachs and possibly cyclic vomiting. In one the vomiting was so persistent that nutritive enemata and saline injections combined with 180 grains of soda bicarbonate were given daily for a week. There was acute congestion of the kidneys, with large hemorrhages of visible blood on two occasions. Albumin and casts were present, but the specific gravity was low, due to the large injections of saline solution by rectum, and quite marked diuresis resulted. Preparatory treatment of cases with acetone in the urine by administration of soda bicarbonate is indicated, and the administration of oatmeal or of glucose may be of value.

It has been the author's object to merely briefly call attention to this interesting problem of acidosis, and to suggest in future a careful examination of the urine for the acetone bodies in all cases.

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[1] Acidosis and Associated Conditions, Arch. Int. Med., 1908, ii, 330.
[2] Jour. Amer. Med. Assoc., Sept. 17, 1910.
[3] N. Y. Med. Jour., June 26, 1909.

## BOTULISM

This is a variety of meat-poisoning due to the Bacillus botulinus, isolated by Van Ermengen from raw ham. The symptoms resembled those of so-called sausage-poisoning. This same type of poisoning has occurred from eating beans that were imperfectly canned. These were probably infected through the manure of pigs. Meat may be extensively infected with the Bacillus botulinus, and contain a large quantity of the toxins, without showing the signs of decomposition. The filtered toxins may produce the effects.

**Incubation.**—The symptoms seldom appear before twelve to twenty-four hours after ingestion of the infected meat. They are: First, disturbances of the external muscles of the eyeball, such as ptosis, abducens paralysis, disturbances of associated movements with nystagmus; second, disturbances of the internal muscles of the eyes, such as enlargement and rigidity of the pupils; third, there are swelling or paralysis of the tongue, pharyngeal and laryngeal paralyses, and disturbances of the heart and respiration; fourth, there may be weakness or paralysis of motion. Changes in sensibility and consciousness usually do not accompany these disturbances. Fever has frequently been absent and there has even been absence of disturbances of the stomach and intestines. This condition is not so rare abroad and occurs in this country.

**Treatment.**—Evacuation of the gastro-intestinal tract. Treatment of symptoms. Herter recommends Kempner's antitoxin when procurable.

## HYDROGEN SULPHID AUTO-INTOXICATION

In health the formation of hydrogen sulphid seems probably limited to the large intestine and a small adjacent portion of the small intestine. Under pathologic conditions it is formed in other parts of the digestive tract. In chronic ectasia hydrogen sulphid is at times liberated when putrefactive changes take place. Herter believes the eructation generally occurs from this viscus, so that no symptoms result. He holds that the stagnating stomach-contents probably go on to the formation of cystin, and that the Bacillus lactis aërogenes, Bacillus coli, and other bacteria produce hydrogen sulphid therefrom. Senator[1] reports a case of auto-intoxication from hydrogen sulphid (hydrothionemia). After an error in diet the patient became ill with gastro-enteric catarrh. On the third day vomiting accompanied with the odor of hydrogen sulphid occurred. The patient became dizzy and went into collapse, without loss of consciousness. The eructation of gas continued, and the first urine gave the reaction of hydrogen sulphid. The case recovered.

Betz[2] and Stefanio and Emminghaus[3] describe cases. Headache, dizziness, delirium, mental depression, drowsiness, stupor, and collapse were prominent symptoms.

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[1] Berlin. klin. Wochenschr., v, p. 254, 1868.

[2] Memorabilien, lx, p. 145, 1864.

[3] Berlin. klin. Wochenschr., lx, pp. 477, 491, 1872.

**Treatment.**—Catarrh of the gastro-intestinal tract, ectasia, and intestinal putrefaction must receive appropriate treatment as required.

## ENTEROGENIC CYANOSIS

This peculiar type of auto-intoxication was first described by Stokvis.[1] The patient, aged fifty-eight, suffered from severe enteritis with pronounced cyanosis of the skin and visible mucous membranes, together with a swelling of the terminal phalanges. Spectroscopic examination of the skin and mucous membranes showed a band corresponding to the absorption spectrum of methemoglobin. It was believed that poison substances were formed in the intestines which transformed part of the hemoglobin into methemoglobin. Talma[2] reported 3 cases. Van der Bergh[3] reported 2 cases, one of which was evidently due to sulphemoglobin (from hydrogen sulphid) in the blood, and in the other the blood contained nitrites.

Van der Bergh also reports a case in a child nine years of age, who suffered from marked digestive disturbances with diarrhea. Anuria was marked. Child was very cyanotic and the ends of the fingers were clubbed; abdominal distention marked. Urine contained no albumin, no sugar, but a little indol. No cardiac affection present.

Several cases of sulphemoglobinemia[4] have been associated with obstinate constipation, the relief of which has been followed by improvement.

*Technic of Clinical Spectroscopic Blood Examination.*—Several cubic centimeters of blood are obtained, preferably from a vein. Examination can be made by drawing a few drops from the ear or finger. The blood should be drawn directly into about twice its volume of distilled water. This lakes the blood and gives a watery solution of the blood pigment. The solution is then filtered several times through a single filter-paper to remove the corpuscles and fibrin. A few drops of this solution are placed in a test-tube or, preferably, in a glass dish or bottle with parallel sides, and examined with the spectroscope by transmitted light. To the specimen distilled water is added drop by drop until the dilution is obtained which allows a good transmission of the red light only. If a black band is seen in the red, the blood contains either methemoglobin or sulphemoglobin. If the band persists on the addition of a few drops of ammonium sulphid, the pigment is sulphemoglobin; if it vanishes, it is methemoglobin. The addition of a reducing agent, as hydrazene or phenylhydrazene, may intensify a sulphemoglobin band which was too faint to be seen otherwise.

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[1] Festsch. f. v. Leyden, l, p. 597, 1902.

[2] Tijdschrift voor Geneesk., li, p. 721, 1902.

[3] Deutsch. Archiv. f. klin. Med., xciii, p. 86, 1905; also Berlin. klin. Wochenschr., No. 1, p. 7, 1906.

[4] Clarke, N. Y. Med. Record, Dec. 3, 1910.

Methemoglobin results from drug poisoning, such as from large quantities of bismuth subnitrate, or from auto-intoxication from absorption of nitrites from the intestines in chronic diarrhea; while sulphemoglobin results from auto-intoxication associated with chronic constipation, the result of hyperformation or hyperabsorption of $H_2S$, or of the presence of an abnormal reducing agent in the blood, acting with a small trace of $H_2S$.

**Treatment.**—This should be directed to the intestinal tract. Proteins should be reduced; sour milks given; purgatives; enteroclysis and the general treatment of intestinal putrefaction by intestinal antiseptics are indicated.

## METEORISM (TYMPANITES)

This is defined as an accumulation of gas in the intestines. Flatulence is used to indicate a great formation of gases that are removed by eructations and flatus.

Part of the gas thus accumulated is expelled and part is absorbed, and thus the volume of gas in the intestines is regulated.

**Etiology.**—The causes of tympanites are as follows:

1. An increased introduction (ingestion) of gas.
2. The development of excessive gas within the intestines.
3. A diminution or impairment of the eliminative power of the intestines for gases.

1. *The increased introduction of gases may be due to the excessive drinking* of aërated beverages or the swallowing of air.

*Arëophagy* (air swallowing) usually occurs in hysteric women, and may result from shock or emotional disturbance. It is generally involuntary, due to spasm of the pharynx. The symptoms are distention after food, loss of appetite, frequent noisy eructations, often insomnia or sleeplessness, constipation. Frequently mucous colic or gastroptosis (enteroptosis) coexist. Rapid deglutition movements precede the eructations.

The mucous colic or enteroptosis should be treated; the hysteria combated; pharyngeal spasm checked by keeping the mouth widely open, applying cocain (1 per cent.) locally, blisters externally, belladonna, bromids, or valerian internally; hypnotic suggestion in some cases; strychnin to stimulate the muscular tone of stomach; food should be concentrated.

2. *Formation of Abnormal Quantity of Gas in the Intestines.*—This may be due to fermentation of the carbohydrates or to putrefaction of the proteins. An excessive amount of fermentable or putrefactive material or food which cannot be assimilated are factors. Catarrhal conditions influence the activity of the ferment and putrefactive organisms.

3. *Diminished Elimination of Gases from the Intestines.*—This is due to mechanical obstruction or to a reduction or inhibition (paralysis) of the muscular power of the intestinal wall.

Among the causes are stenosis, intestinal obstruction, paresis of the intestines in the infectious diseases, such as typhoid fever, pneumonia, cerebrospinal meningitis, peritonitis, etc.

In pathologic conditions, where there is circulatory disturbance in the intestinal walls, as in peritonitis, etc., absorption of gases must be interfered with. It is difficult to estimate to what degree this is a factor.

*Nervous Meteorism.*—This is most common in the hysteric, and usually occurs as a diffuse distention of the abdomen (tympanites hystericus), but also as a circumscribed swelling (phantom tumor).

Various factors have been suggested, such as swallowed air; insufficiency of the pylorus, allowing passage of air from the stomach to the bowels; or continuous contraction of the diaphragm (Talma).

With narcosis the abdomen becomes flaccid, while with returning consciousness meteorism recurs, and air cannot be detected entering or leaving the tract, hence Talma denies that there is any increase of gas in the intestines. Some attribute the condition to acute general paresis, of sudden onset, passing off rapidly, analogous to other hysteric paralyses.

**Symptoms.**—The shape of the abdomen is usually altered, a feeling of tension is almost always experienced. With general meteorism the abdomen is distended quite symmetrically, while with partial distention the affected parts stand out in marked relief.

With local distention the coexistence of visible peristalsis, as with stenosis, simplifies the diagnosis. The degree of distention generally corresponds to the amount of gas present; but the weaker the muscles and the more reduced their tone, the greater is distention. An example of this is marked tympanites in peritonitis.

When the abdominal muscles are tense sometimes the diaphragm, heart, and lungs are forced upward, and severe dyspnea results, which occasionally may be followed by a fatal issue.

In severe cases there is a constant feeling of pressure and a desire to pass wind, while colicky pains are sometimes present. As a rule, no flatus is passed, or in inconsiderable amounts at long intervals.

Palpation shows the abdominal walls are very tense. They present an air-cushion resistance. The percussion note is abnormally low and loud, the tympanitic ring is usually lost. With auscultatory percussion a metallic sound is elicited. The liver dulness may be reduced and finally disappear. There are some cases of meteorism, in which the wind is passed almost constantly from the anus with considerable noise. Rosenheim believes, as these gases are odorless, that the air is pumped into the rectum and emitted again as flatus.

**Prognosis.**—If occlusion of the intestines is present, the outlook is serious. Ordinary cases are favorable.

**Treatment.**—This should be directed toward removal of the cause responsible for meteorism, such as peritonitis, typhoid, stenosis, etc.

Drinks and foods known to produce flatulence must be prohibited. Avoid carbonated waters, beer, champagne, cider, excessive carbohydrates, rich pastry, etc.

Intestinal fermentation or intestinal putrefaction, if they cause the meteorism, must he treated after the methods already described.

Among the intestinal antiseptics are ichthalbin, ichthoform, formidin, salol, salicylate of soda, benzonaphtol, urotropin, and sodium benzoate, average dose of each, 5 grains (0.3) t. i. d. after meals. They may be given in shellacked capsules. Bismuth salicylate and bismuth subnitrate, 5 to 10 grains (0.3–0.6) t. i. d., are of service.

Calcined magnesia, lime-water, and charcoal are suggested to absorb the gas.

In mild forms of flatulency various carminatives have been employed, such as caraway seed, peppermint, mint, thyme, cinnamon, cloves, anise seed, nutmeg, sassafras, and fennel. These are best given as infusions. Asafetida and the oil of cloves are believed by Brunton to aid absorption of $CO_2$ and $H_2S$.

Cathartics and laxatives may be necessary when there is no peritonitis or obstruction. Physostigmin salicylate, $\frac{1}{100}$ to $\frac{1}{50}$ grain (0.0006–0.001), has been recommended in severe cases.

Massage, abdominal douches, electricity, and electric enteroclysis are of service in cases where there are no anatomic lesions. Friction of the abdomen with spiritous, aromatic, or ethereal substances, such as camphorated oil, turpentine, oil of cajeput, etc., are serviceable.

The introduction of a colon-tube may aid the escape of gas. Water enemata of soapsuds, containing spirits of menth. piperit., 1 dram (4.0), or oil of turpentine, 1 dram (4.0), are useful.

Puncturing the intestines with a trocar is a dangerous procedure.

Meteorism in the hysteric occasionally disappears without treatment. Attention should be paid to the nervous condition. Valerian and asafetida are useful by mouth or enema in such cases.

Pill asafet., one t. i. d.; emulsion (milk) asafet., 1 to 2 ounces (30.0–60.0), by enema.

Zinc valer., 2 grains (0.125), or ammon. valer., 5 grains (0.3), t. i. d.

Tinct. valer. with tinct. lavend. co., *āā* 2 ounces (60.0). Dose, 1 dram (4.0) in water t. i. d. after food. Iron, arsenic, and strychnin are of tonic value in the nervous cases.

Massage, friction, and a tight abdominal band are useful.

## INTESTINAL PAIN (INTESTINAL COLIC; ENTERALGIA)

Intestinal pain can be distinguished as follows:

1. Pain originating from inflammation of the intestinal wall or of its peritoneal coat.
2. Pain of colic.
3. Nervous enteralgia, described under Neuroses.

**Colic** is the painful stimulation of the intestinal nerves which is produced by severe tonic contractions of the intestines. G. F. Shiels[1] holds that the pain is produced in the peritoneal coat. Kast and Meltzer have demonstrated by a series of experiments that the intestines are sensitive to pain.

**Etiology.**—Organic lesions of the intestines, excess in or improper articles of food, cold drinks, substances causing marked gas formations, fecal accumulation, intestinal worms, foreign bodies, gallstones, enteroliths, tainted foods, large quantities of mucus, as in mucous colic, exposure to cold, gout, occasionally ulcers, internal strangulation of the bowels, stenosis, purgatives, lead- and copper-poisoning may produce colic.

**Symptoms.**—The pain of colic is peculiar—pinching, boring, or occasionally of a tearing character—it occurs in paroxysms which may last a few seconds or be prolonged several hours. It usually appears suddenly and disappears as rapidly. Its intensity may be so severe that fainting and collapse occur.

If colic begins from error in diet, there may be gastric disturbances, belching, nausea, and vomiting. Obstinate constipation and flatulence may be present, or if the cause be from improper food, diarrhea. The pain frequently starts at the umbilicus and remains localized or radiates in other directions. The face of the patient shows his suffering. Pressure over the abdomen relieves the pain in some, while in others it increases it. There may be straining sensations in the bladder and rectum and occasionally borborygmi can be heard. Peristaltic movements can be seen in thin patients. If accumulated fecal masses and gas are evacuated spontaneously or by injections, the attack rapidly subsides.

Spastic contractions of the intestines may be encountered. If they involve a large part of the bowels, as in lead-colic, the abdomen appears trough shaped. In stercoral and wind-colic, it is usually tympanitic.

**Diagnosis.**—In enteralgia due to anatomic lesions of the intestines the pain is increased by pressure. There is frequently diarrhea and the stools contain blood, mucus, and, rarely, pus.

With rheumatism of the abdominal muscles the pain is superficial and not within the abdominal cavity. It often changes its location. Pressure increases the pain, while rest and antirheumatic medicines diminish it.

With peritonitis there are fever, tenderness on pressure, muscular rigidity, meteorism, absence of abdominal respiration, increase of polynuclears, and leukocytosis.

Hyperesthesia of the abdominal wall usually occurs in hysteria and neurasthenia. The pains are superficial, lying chiefly in the skin. The faradic current often removes the pain. Biliary and renal colic are recognized by the location of the pain and characteristic symptoms. Neuralgic pains are superficial and radiate.

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[1] Amer. Jour. of Surgery, April, 1908.

**Prognosis.**—These cases end in recovery, with rare exceptions.

**Treatment.**—For the relief of pain, morphin by hypodermic, $\frac{1}{8}$ to $\frac{1}{4}$ grain (0.008–0.016), or codein, the same or even a double dose, or tincture of opium, 10 minims (0.59 c.c.), heat to the abdomen, and hot saline enema or enteroclysis at 115° F. Squibb's mixture is useful.

Later, the bowels should be thoroughly evacuated by an enema, the simple soapsuds enema, 1 quart (liter), or which may contain $\frac{1}{2}$ pint (250 c.c.) of olive oil and 1 dram (4.0) of spirits of turpentine. A cathartic, castor oil, $1\frac{1}{2}$ ounces (45.0), or calomel, 5 grains (0.3), followed by a saline cathartic, should be given.

The cause of the colic should be corrected, as should any errors in diet. Fluid diet should be employed.

Tincture of belladonna, 5 to 10 minims (0.3–0.6), the writer finds of value.

## VISCERAL ARTERIOSCLEROSIS

Harlow Brooks[1] calls to our attention that the presence of diminished visceral function, with occasional and otherwise unaccountable elevations of the blood-pressure, should, particularly in connection with possible etiologic factors, lead to a suspicion of some localized, if not general, area of arterial disease. When associated with pain of peculiar anginal character, location in some special organ may be within the range of possibility. I feel quite certain that careful observation of cases of obscure abdominal pain, paroxysmal in character and associated with elevation of the blood-pressure, will eventually make it possible to diagnose these generally unrecognized types of arterial disease while in a stage when something may still be done for their alleviation or for the prevention of their further progress.

Some of these patients give a history of nervousness, gastric catarrh, and pain; *vomiting* and even hematemesis may occur. Meteorism and constipation are not uncommon. Pain over the pancreas and intestinal functional disturbances have been noted. When such symptoms occur in a patient with well-marked arteriosclerosis, and are relieved by iodids and nitrates, one is justified in diagnosing abdominal arteriosclerosis. The determination of increased blood-pressure by means of the sphygmomanometer aids the diagnosis of the milder types of such cases. This method should always be employed. Under Indicanuria the writer reports an interesting case.

**Etiology.**—Alcohol, nephritis, syphilis, old age, tuberculosis, tobacco excesses, and toxemia are the chief causes.

Perutz[2] shows we must differentiate between angina pectoris and angina abdominis due to arteriosclerosis:

I have recently seen several cases who have complained of pain in the epigastrium, belching, constipation, and headache. The gastric analysis showed deficient hydrochloric acid secretion and the pulse the characteristics of arteriosclerosis. Treatment afforded no relief until the nitrites were administered. "Gilbride[1] reports a number of cases with symptoms of pain in the epigastric or umbilical regions, rarely in the lower abdomen. This is increased by exertion and in some cases during digestion. Weakness, loss of weight, abdominal distention, and belching are present. Bowels are constipated or there is constipation alternating with diarrhea, or occasionally normal movements. There may be vertigo or disturbances of vision. The gastric analysis showed in most cases deficiency of hydrochloric acid; in one it was nearly absent and in one normal. Stomach motility normal or increased. Lactic acid and Boas-Oppler bacilli were absent. Cancer had been suspected. The radial pulse and blood-pressure may show arteriosclerosis. In some cases there are no evidences of this, but there are two symptoms significant of abdominal arteriosclerosis—*tenderness of the abdominal aorta* with *epigastric pulsation*. Some patients may have attacks of angina pectoris with pain referred to the epigastrium. The age of the patient is usually over forty." Ortner[2] has contributed to the subject, demonstrating the disturbances of the motor, secretory, and absorptive functions of the intestines due to arteriosclerosis; and Akin[3] has reported several cases. Nitrites afford the most relief, with the addition of potassium or sodium iodid. Tincture aconite, 5 to 10 drops t. i. d., aid in lowering excessive high tension.

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[1] Amer. Jour. Med. Sci.

[2] Münch. Med. Wochenschr., May 28 and June 4, 1907.

## SENILE DYSPEPSIA

The dyspepsia of old age has been investigated by Fenwick,[4] who shows that in every 100 cases of chronic dyspepsia in persons over sixty-five years of age, 66 are secondary to organic diseases of some important organs of the body, while 34 owe their symptoms to a progressive degeneration of the secretory structures of the stomach and intestines. Disease of the kidneys, heart, lungs, liver, or a gouty condition all have a bearing in a large percentage of cases on the gastro-intestinal disturbances of the aged, and in a certain number gastric hypersecretion of long duration resulting from an old gastric or duodenal ulcer may be a factor. Chronic appendicitis or fibroid degeneration of the appendix may also result in gastro-intestinal disturbances.

In about one-third of the cases no organic change can be located in any particular organ. There is, however, an impairment of the secretory functions of the digestive tract, the general assimilative powers decline, the body assimilates less organic matter, and destroys less inorganic material with a resulting waste of tissue in many cases. In others disturbances of metabolism occur, which result in obesity and in the derangement of the eliminative functions. In such there is associated frequently fatty degeneration of the organs.

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[1] Gastro-intestinal Disturbances Due to Arteriosclerosis, Jour. Amer. Med. Assoc., March 20, 1909. [2] Volkmann, Samml. klin. Vortr., No. 347.

[3] Jour. Amer. Med. Assoc., June 5, 1909.

[4] London Lancet., Nov. 6, 1909.

Even though there be no marked evidences of general arteriosclerosis, the author believes these disturbances of the gastro-intestinal functions in the aged to be in most cases a manifestation of visceral arteriosclerosis, varying from a mild to a severe type, with resulting damage to the glands presiding over the functions of digestion.

Undoubtedly, the condition of the mouth, deficiency or absence of teeth, decayed roots, with associated putrefaction in the mouth, all have an influence. Insufficient mastication also has a bearing on the subject.

**Gastric Analysis.**—Examination of the gastric contents after the test-breakfast shows a diminution of free hydrochloric acid (hypochlorhydria), and sometimes even achylia gastrica.

**Intestinal Functions.**—The functions of the intestinal digestion should be tested. Intestinal fermentation or intestinal putrefaction are present.

**Symptoms.**—Belching of gas and flatulence occur. Constipation is usually present, though in some it alternates with a diarrhea. Many cases suffer from loss of appetite and from insufficient nutrition and there is loss *of weight as a result.*

Other patients are of a different type and are obese. They suffer from belching of gas and flatulence. Their appetite is good, in fact, cannot be satisfied, but elimination is imperfect. The stomach is atonic, but pyloric insufficiency is often present. There is atonic constipation, but it frequently alternates with lienteric diarrhea.

The first class of patients, the asthenic type, Niles[1] classifies as hyperkoric dyspepsia, in whom there is early satiety for food, loss of appetite, or even sitophobia. As a result scarcely sufficient food is ingested to properly support life.

The second class, the obese or sthenic type, he characterizes as akoric dyspepsia, since they are never satiated.

One also meets among the aged cases with very marked symptoms such as described under Visceral Arteriosclerosis.

**Diagnosis.**—The diagnosis can be made by exclusion of other factors, taken in association with the age of the patient. The determination of increased blood-pressure by means of the sphygmomanometer is also of value. It should always be employed.

**Treatment.**—The mouth should be cleansed with boric acid, glycothymolin, listerin, borolyptol, or some mild antiseptic several times daily. Old roots and decayed teeth should be extracted and artificial teeth should be worn, so that the patient can masticate properly. Active catharsis should be avoided; simple remedies, such as purgen, cascara, phenolax, regulin, and small enemata of olive oil, are indicated. Dilute hydrochloric acid with nux vomica should be given for the subacidity. A convenient preparation is oxyntin with nux vomica in capsules, 5 to 10 grains (0.3–0.5) t. i. d., before or after meals. Taka-diastase, 5 grains (0.3) t. i. d., is valuable to aid starch digestion, and holadin, 5 to 10 grains (0.3–0.5) t. i. d., or some other pancreatic preparation for intestinal dyspepsia.

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[1] Amer. Jour. Med. Sci., Jan., 1911.

Other remedies are noted under the section on that subject. The nitrites, nitroglycerin, $\frac{1}{100}$ to $\frac{1}{50}$ grain t. i. d., or more frequently, should be administered for the arterial condition, and the iodids may be added, sodium iodid, 5 grains (0.3) t. i. d.; preferably, as it causes less disturbance of the stomach.

*Exercise.*—Those cases characterized by malnutrition should take a minimum amount, but be in the fresh air a much as possible, while activity is indicated for the obese within the limits of fatigue.

*Diet.*—Food should be prepared in the most digestible form. It is preferable to administer four or five small meals daily to three large ones. In the asthenic cases with malnutrition, food of a high calorie value, such as fats and starches carefully prepared, are indicated, while patients of obese type should receive a minimum of these. In these cases the frequent small meals relieve best the sensation of hunger. The above rules are only general.

As the condition is one of deficient hydrochloric acid secretion, the diet can be much the same as for achylia gastrica.

In the earlier stage of treatment peptonized milk may be given for a brief period. Matzoon, koumiss, lactone-buttermilk, bacillac, eggs, and barley and rice gruel. Excessive fluids, however, tend to produce too much gas in some patients.

Small quantities of thicker gruels and soups, such as pea, strained bean, potato, barley, rice, and sago often agree. Fenwick holds that barley, oatmeal, and rice must be given with caution. Much depends, however, on the cooking of the cereal, which must be thorough, and it should be passed through a colander so to ensure removal of the membranous coverings.

As the patient improves, meat should be given in small quantities and finely minced, such as fish, chicken, game, calves' brains, sweetbread, calves' feet, rare or raw scraped beef, and pigeon.

Fats, such as butter or cream, must be tried on the sthenic cases suffering from malnutrition. They sometimes agree and at times not. The diet must be necessarily modified by the conditions found in the functions of the intestine, which as noted should be determined. Red meat should be avoided when there is intestinal putrefaction of a marked character, and the condition should receive treatment. Toast baked in the oven to dry it out and zwieback are preferable to bread. Crackers are admissible, green vegetables and oranges may be tried. If plain milk disagree and peptonized milk be disagreeable, a little lime-water or citrate of soda may be added to the milk.

Flavored sanatogen, tropon, or somatose may be added to the milk or gruels.

## ANOMALIES IN THE POSITION AND FORM OF THE INTESTINES; ENTEROPTOSIS

Various anomalies in the position and form of the intestines occur, in some cases congenital and in others acquired, of which Koch and Curshman have made a study.

They may be congenital in some cases, acquired through coprostasis, the weight of a tumor, or the formation of adhesions, in the majority of cases the colon being affected. Various angulations of the sigmoid, especially due to adhesions, are described by J. P. Tuttle. Inflation with air and the use of bismuth injections with the $x$-rays aid diagnosis.

**Hirschsprung's disease,** or congenital primary dilatation and hypertrophy of the colon, is found at birth. Dyspeptic symptoms, obstinate constipation, and colicky pains with great distention are present. By insertion of a finger or catheter into the rectum a movement will occur with relief of the distention. Enteroclysis, electricity, etc., are of value.

Idiopathic dilatation of the colon is a rare event, and enteroptosis, the most common displacement, is described under Gastroptosis.

Idiopathic dilatation of the colon, in which there is no mechanical obstruction to the feces, may be congenital, develop early or later after birth, or be acquired in adult life. Nervous derangement, habitual constipation, rickets, or chronic colitis may be factors. The colon and especially the sigmoid are greatly distended, so that the protrusion of the abdomen may justify the term " balloon man." Constipation or irregularity of the bowels, gaseous distention, intestinal putrefaction, shortness of breath, etc., occur. Enteroclysis, regulation of the bowels, diet, and treatment of indicanuria are indicated. Abdominal support is of service. Rarely, operative procedure is required.

## INTESTINAL SAND

This material is gritty, contains organic matter, inorganic salts, especially calcium phosphate and carbonate, but *no cholesterin*, which distinguishes it from biliary sand.

It has been considered by some as a manifestation of the arthritic diathesis. It is usually associated with mucous colic or possibly constipation. It occurs generally in women between thirty and forty years of age.

The associated condition must be treated. Sodium bicarbonate and bismuth salicylate have been recommended.

False intestinal sand, such as the residue of vegetable food, especially from *pears*, must not be mistaken for true sand. Symptoms are absent.

## CHAPTER XXII

## INFECTIONS BY THE BACILLUS COLI

INVASION of the kidneys by the colon bacillus is not a particularly uncommon occurrence. As the author[1] has diagnosed this condition as a serious complication in a number of cases of typhoid fever, and as he has furthermore observed the invasion of other organs by the colon bacillus, it seems proper, in view of its source, that this infection should be described in this volume. The colon bacillus has, furthermore, a definite relation to certain infections in the gastro-intestinal tract. For example, it has been found as a factor in acute appendicitis and also in the fluids of acute peritonitis. It has been responsible for inflammation of the pancreas, for gall-bladder inflammation, and colon bacilli have been found as the nucleus of gall-stones and pancreatic calculi.

Furthermore, Fenton B. Türck has fed colon bacilli to dogs and has experimentally produced gastric ulcer in these animals.

Reference has been made to Herter's investigations into intestinal putrefaction as to the influence of the Bacillus aërogenes capsulatus in the production of pernicious anemia. The possibility that this last condition may at times be the result of colon bacilli infection is suggested by A. Charlton[2] in his researches at McGill University in an article entitled On the Anemia Produced by Repeated Injections of Cultures of a Colon Bacillus of Low Virulence. Striking results followed intravenous injections—the experiments being carried on for many weeks.

They were as follows: Advanced anemia occurred, in some respects strikingly like the conditions found in pernicious anemia, namely, a great diminution in the number of red cells, marked poikilocytosis, the appearance of pear-shaped cells, crescents, macrocytes, microcytes, and, lastly, nucleated red cells. On suspension of the injections for some weeks, even when the fall of erythrocytes was from 5,500,00 to only 1,500,000, spontaneous recovery took place. When injection of the bacilli was resumed, the injurious results were more marked than before. But in addition to the effects on the blood, strikingly progressive changes were produced in the spinal cord, consisting of diffuse degeneration in the columns of Goll, and in the anterolateral columns of the cord, closely resembling the conditions occurring in the spinal cord in some cases of pernicious anemia; extreme emaciation occurred—the animals dying apparently from pure asthenia, though they fed well and suffered from no gastro-intestinal disturbances. Wm. H. Thomson[1] compares this condition to progressive anemia with emaciation and nervous weakness, grouped under the vague term neurasthenia.

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[1] Boston Med. and Surg. Jour., Nov. 30, 1911.

[2] Jour. of Med. Research, vol. viii, No. 2, Nov., 1902.

H. G. Harris[2] of New York has reported an interesting case of *infantile paralysis* in which colon bacilli were found in large numbers in the urine. The patient recovered under the use of urotropin. In view of the changes produced in the spinal cord experimentally by the injection of colon bacilli into the circulation, the clinical case cited is of interest.

**Method of Infection by the Colon Bacillus.**—Some suggest that the origin of colon bacilluria is due generally to the *introduction of these organisms from without* (*ascending infection*). Thus, catheterization is given as a cause. Box holds that he has found this condition more frequently among girls than boys, and explains it by the short urethra in the former, which more readily allows entrance of the bacilli. He states there is no more *common cause of enuresis among children* than the presence of this bacillus. While doubtless these organisms may enter the bladder through the urethra instead of descending from the infected kidneys, it is difficult to account for the persistent enormous multiplication of these bacilli in the urine, when, unlike all urines decomposed by other bacteria, in *colon bacillus* cases it remains strongly acid and not alkaline, and gives off no ammoniacal odor. Of course, in all cases of organs infected other than those of the urinary tract, the bacilli must come through the blood, and it is the author's belief that in most cases the bacilluria results from auto-infection from the intestinal tract—in some cases the bacilli causing an active process in the kidneys—while in others a few bacilli are eliminated through these organs without clinical symptoms for a considerable period. As a rule, there is some ulcerative[3] process in the intestinal canal, or in some cases probably a mere solution of continuity. Chronic constipation alone may suffice to allow the occurrence of chronic infection from the colon. The possibility of entrance through the tonsils has been suggested.

In fact, two such cases have been reported. Infection by *contiguity* may also occur, as by rupture of an abscess, and infection through the lymphatic system may also be possible. *Hematogenous infection*—*i. e.*, through the blood—is the most common method.

**Types of Infection Produced by the Colon Bacillus.**—Among the types of infection produced by the colon bacillus are acute, subacute, and chronic nephritis, pyelitis, pyelonephritis, septic infarcts of the kidney, cystitis, and enuresis. Cases of aggravated constipation which exhibit typhoidal symptoms are occasionally met with in which isolation from the urine and blood of the Bacillus coli communis, or positive and exclusive agglutination of the patient's serum against most strains of this organism prove these cases to be a general infection with the colon bacillus. An epidemic of this type has been reported in Bavaria.[1] Phlegmonous enteritis (colon infection) is recorded.

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[1] Med. Record, May 28, 1910.

[2] N. Y. Med. Jour., April 1, 1911.

[3] W. H. Thomson, Acute, Subacute, and Chronic Infection of the Kidneys and of other Organs by the Bacillus Coli, Med. Rec., May 28, 1910.

Attacks of intermittent fever, the fever coinciding with the appearance of colon bacilli in the urine, and the intermission, with their disappearance, have been reported. Intra-abdominal exudates and tumors, believed to be tubercular or cancerous, have been demonstrated to be pure colon bacilli infections. Cases simulating pernicious anemia may be due to this infection, and choroiditis has also been reported as due to the same cause. Gastric ulcer and achlorhydria hæmorrhagica gastrica secondary to a primary appendicitis infected by the colon bacillus are also possibilities. The author reports in this article a case of a private patient at the New York hospital, who was under his care, who suffered from general infection with the colon bacillus. This patient had a double pyelitis, cystitis, double pneumonia, purulent bronchitis, two attacks of acute colitis, and myocarditis, all in succession, and ultimately recovered. Colon bacilli were present in the urine and sputum in enormous numbers. In acute pancreatic disease and acute cholecystitis this type of infection must be considered, and in cases of acute postoperative general hemorrhagic attacks in patients with gall-stones and pancreatitis.

Subdiaphragmatic abscess and wound infection by colon bacilli may also occur. Reference has previously been made to the fact that colon bacilli are found as a nucleus of gall-stones and pancreatic calculi, and to Harris' case of apparent infantile paralysis with colon bacilli in the urine, cured by the administration of urotropin. Experiments have also demonstrated the possibility that colon bacilli may be factors in the production of cirrhosis of the liver.

**Reaction of the Urine in Colon Bacillus Infection.**—The urine reaction is *acid* in *colon bacillus infection*. Some claim it is at times alkaline, but the writer has not found this so unless after the administration of large quantities of water or of alkalis.

**Symptoms of Colon Bacillus Infections.**—On account of the diversity of these infections, it seems best to the author to illustrate them by various cases. Acute infection of the kidneys will be first described. An interesting case is reported by Wm. H. Thomson[2] in consultation with Robert Abbé:

"On the fifth day after operation for pelvic tumor, the patient complained of cramps and pain in the abdomen extending to the rectum. These were accompanied by nausea and frequent vomiting, followed by a rise of temperature to 104.3° F. The patient then became drowsy and apathetic, with muscular twitchings in the face and upper extremities. The wound was healthy and healed by first intention. These symptoms cleared up. On the twenty-second day after operation the patient had slight chills lasting twenty minutes, rise of temperature to 103.5° F., with the development of the symptoms described above. In addition the pulse became rapid, small, and irregular. The chills, sudden onset, early cerebral symptoms, which may pass into coma or delirium, but no convulsions, rapid rise of temperature, and some vomiting, Thomson believes pathognomonic of involvement of the kidneys by some septic organism, of which the colon bacillus is the most common. The following day the urine became loaded with pus, blood, and casts, and colon bacilli were found in large numbers in the urine.

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[1] De Haan and De Gouge: Laboratte Weldobrecken Mederlingen, Bavaria, 1902.

[2] Med. Rec., May 28, 1910.

"On account of the vomiting, urotropin and sodium benzoate, each 10 grains, were administered by rectum every three hours. As the patient also had oliguria (16 to 26 ounces daily), enteroclysis with Kemp's tube was ordered A. M. and P. M., 2 gallons of normal saline solution at 115° F.; the urine increased to from 60 to 112 ounces in twenty-four hours. During the course of a week the pus and blood cleared up. The urine continued acid with a specific gravity averaging 1008, but it still showed a diffused white cloud. This is characteristic and is caused by immense numbers of colon bacilli in suspension. The cerebral symptoms and vomiting ceased within twenty-four hours after initial treatment, and the temperature fell to 99° F., and thence to normal. The bacilluria still persisted until on the twentieth day she developed symptoms of subacute appendicitis. Abbé operated under local anesthesia, and removed a diseased appendix with several hemorrhagic points in its lumen and a large ulcer in its base at the junction with the cecum. The bacilli subsequently disappeared from the urine. Though the treatment alleviated the severe symptoms, the focus of infection was present until removed." Chas. R. Box, of St. Thomas Hospital, London, in an article in the Lancet[1] observes that in many *cases where the kidneys are involved* in *typhoid fever, scarlet fever, measles, and diphtheria,* the organism found in the urine is the *colon bacillus.*

In view of this fact, the colon bacillus should be examined for in all cases. In any event, even if not found, it would seem to the author that the large doses of urotropin and sodium benzoate are the best disinfectants for the kidneys.

Wm. H. Thomson[2] reports an interesting case of A. Seibert, of New York, during the course of scarlatina. Dr. Seibert found the girl, age eleven, in the sixth week of her illness, suffering from universal edema, most marked in the face, some ascites, pulse 48, intermitting every third beat. She was semiconscious, with total blindness, which had persisted for five days, nausea, and incessant vomiting, which had lasted for ten days, necessitating rectal feeding. Two severe uremic convulsions had occurred on the previous day. Only

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[1] Jan. 11, 1908.

[2] Med. Rec., March 21, 1908; May 28, 1910.

4 or 5 ounces of urine had been passed in the previous twenty-four hours, and this contained but a moderate amount of albumin. Urotropin, 5 grains dissolved in 1 ounce of cold water, with 15 drops of dilute hydrochloric acid, was administed and retained. The same dose of urotropin was continued every three hours. The headache first disappeared, marked diuresis set in, so that 110 ounces of urine were passed in the next twenty-four hours, and all the edema disappeared within forty-eight hours. The amaurosis diminished in two days and, finally, disappeared in a week, after which she made an uninterrupted recovery.

A second illustrative case is that of a physician who had been treated for chronic ulcerative colitis and who had had a rectal ulcer excised. Shortly after this he became delirious, with moderate temperatue. This condition lasted four weeks. The urine was examined daily by an expert, who reported blood, albumin, and many casts—laboratory diagnosis, acute parenchymatous nephritis. Thomson, on being called in consultation, finding that the patient had no edema, that the pulse was soft, and the delirium prolonged and active, which were not common in parenchymatous nephritis—also being cognizant of the history of intestinal ulceration —diagnosed infection of the kidneys with the colon bacillus, and requested examination of urine for the same. Meanwhile his usual treatment, urotropin and sodium benzoate, each 10 grains every three hours, was ordered. It was first given by rectum. The laboratory report shows colon bacilli in abundance. Two days later the blood, albumin, and casts disappeared, and six months later the physician reported sixteen hours professional work a day.

**Typhoid Fever.**—Acute invasion of the kidneys by the bacillus coli is not uncommon, and is at times a fatal complication of typhoid fever, especially in the later stages of extensive ulceration. Sudden severe rigors occurring late in the disease, increased albuminuria and casts, often with diminished urine, and at times with commencing delirium, are all significant of colon bacillus infection. The urine should at once be examined for the bacilli, and the usual treatment, urotropin and sodium benzoate, instituted. The author has had numerous such cases during the past few years.

**Pyelitis.**—Acute invasion of the kidneys may be productive of a simple pyelitis, usually bilateral, attacking first one and, after subsidence of the acute symptoms, then the other organ.

There is then a subsidence of symptoms, but recurrences take place usually in one kidney, and these continue until final cure. There is a sudden rise of temperature, 103° to 105° F., often preceded by a chill; first one, and later, frequently the other kidney, becomes tender on pressure. There is pain in the lumbar region, urine is highly acid, contains pus, occasionally blood, and is loaded with colon bacilli. The pyelonephritis of pregnancy, particularly postpartum, is quite frequently the result of colon bacillus infection.

L. C. Dudgeon,[1] in his Erasmus Wilson lecture on Acute and Chronic Infections of the Urinary Tract, also describes such cases. He reports that such attacks sometimes occur with appendicitis. Finding such cases prone to relapse, he recommends treatment with vaccines. As a matter of routine, the author believes that in all cases of pyelitis one should examine the urine for colon bacilli. The dose of urotropin usually given is too small, as will be described under Treatment. If appendicitis is present, as in some of Dudgeon's cases, it should be removed, as it is the source of infection under such conditions.

**Acute Unilateral Septic Infarcts of the Kidney.**—George E. Brewer[2] reports several cases in which the lesions were confined to one kidney. This condition may be due to pure cultures of the staphylococcus pyogenes aureus, virulent forms of streptococci, and in one case the organism was the Bacillus coli. Other authors mention the latter as a frequent cause of this condition. The symptoms, which are sudden in their onset, are chills, a temperature rising rapidly to 103° to 105° F., to even 106° F., tenderness over the kidney, especially at the costovertebral angle (Brewer's point); pus, albumin, casts, and, at times, blood are present in the urine, and colon bacilli when such are the cause of the infection.

The cause of unilateral infarctions can be readily explained by the probable occurrence of preceding lesions in this organ—according to the principle illustrated by S. J. Meltzer—who, on producing minute lesions with long needles in the liver, kidneys, and other organs of rabbits, and then injecting a virulent culture in the vein of the ear, produced abscesses only at the seat of these traumatisms. Infarctions occasionally occur in both kidneys, so that differential urethral catheterization will absolutely determine the correctness of the diagnosis as to whether one or both are involved.

**Chronic Interstitial Nephritis.**—Wm. H. Thomson holds that this condition may be primarily caused by slow infection of the kidneys by the Bacillus coli. Sudden terminations of cases of chronic interstitial nephritis, characterized by oliguria or suppression of urine, are nearly always preceded by acute indigestion in some form, and Thomson always dreads an attack of so-called cholera morbus in an elderly patient, and at once examines the urine.

These cases are frequently attributed to an acute exacerbation of the chronic kidney disease, whereas numbers of such cases have been saved by immediate resort to urotropin and sodium benzoate, administered by rectum when the patients were comatose. He holds that it is suggestive how soon in the course of chronic interstitial nephritis symptoms of increased kidney affection develop after errors of diet. The author, under Indicanuria, has called attention to the acute kidney disturbance produced by intestinal putrefaction, and also noted the value of urotropin and sodium benzoate for the treatment of this condition.

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[1] The Lancet, Feb. 29, 1908.

[2] Surgery, Gynecology, and Obstetrics, vol. ii, No. 5.

**Cholecystitis.**—Inflammation of the gall-bladder is frequently produced by the colon bacillus, which may reach it, as it does probably most frequently through the gall-duct, or, at times, through the blood-stream or lymphatics.

At the Johns Hopkins Hospital the gall-bladders of animals were infected with the colon bacillus, and subsequently cured or inflammation prevented by the administration of urotropin. It would seem wise to examine the urine for colon bacilli in all cases, and in any event to administer urotropin to disinfect the gall-bladder.

H. Batty Shaw[1] gives the histories of 7 cases of his own of colon bacilli infection, in which he states that clinically such patients may be supposed to be sufferers from malaria, tuberculosis, mucous colitis, recurrent cystitis, dyspepsia, acute suppression of urine, typhoid fever, or pernicious anemia. He recommends that every case of chronic febrile affection of obscure origin should have the urine examined for the Bacillus coli.

The author has recently treated a case of chronic pancreatitis with marked steatorrhea and colon bacilluria. In view of an old history of intestinal ulcer, the possible influence of colon bacillus infection in the production of chronic pancreatitis is suggested.

**Choroiditis.**—Robert T. Morris[2] reports the case of a patient referred to him by John S. Kirchendall, of Ithaca, who suffered from choroiditis, which threatened to end in blindness. Morris found that for years the patient had suffered from intestinal troubles due to fibroid degeneration of the appendix, and on its removal the patient recovered, both from the intestinal dyspepsia and also from the choroiditis. Kirchendall states to Morris that Stockton, of Buffalo, has for years asserted that the colon bacillus was the cause of many cases of choroiditis.

**Pelvic Exudate and Intra-abdominal Tumors Due to Colon Bacillus Infection.**—F. Charlton Briseos,[3] Physician to Kings College Hospital, London, relates several instances of the matting together of the pelvic viscera by an exudate, showing caseous changes, which led to a mistaken clinical diagnosis of tubercular nature, but which proved, on microscopic examination, to be caused by the Bacillus coli. In each case colon bacilli were found abundantly in the urine.

*Case 1.*—This patient was a woman with rectal ulcer and chronic hemorrhoids, who gradually lost weight, with daily fever and constant pain in the right flank. These were evidences of an intra-abdominal mass. On laparotomy, the pelvic organs were found matted together, the uterus, posterior wall of the bladder, and a coil of the small intestine being bound together. Under treatment by vaccines she fully recovered.

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[1] Clinical Jour., Feb. 12, 1908.

[2] N. Y. Med. Jour., Jan. 1, 1910.

[3] The Lancet, Oct. 30, 1909.

*Case 2.*—This patient suffered from intra-abdominal tumor, with temperature. The condition was thought to be tubercular. On laparotomy, a large mass of caseous material was found attached to the small intestine, great omentum, left ovary, and tube. This was thought to be surely tuberculous, but no tubercle bacilli were found. There was a structureless material from which pure cultures of colon bacilli were obtained.

*Case 3.*—In an Italian woman, age fifty-two, there was a large tumor which was suspected to be malignant. On laparotomy, it was found to be attached to the wall of the bladder and extended from the pelvis to the umbilicus. It was of dark red color and covered with peritoneum. There were many large blood-vessels passing over its surface. On the supposition that it was malignant and inoperable, it was let alone. For three days after the temperature rose daily to 103° F., and on the fourth day she passed a considerable quantity of pus and large flakes of lymph in the urine. The tumor was found to have disappeared, but from time to time it re-formed and emptied itself, eventually disappearing entirely. From the urine a pure culture of Bacillus coli was obtained.

Briscoe further refers to an interesting number of cases who have attacks of intermittent fever, the temperature coinciding with the appearance of colon bacilli in the urine, and the intermissions, with their disappearance. He advises that the urine be examined *for colon bacillus in all cases of febrile attacks of obscure origin.* He also notes how frequently ulcers in the rectum occur previous to invasion of the kidneys and other organs. He, together with other authors, comments on the pale color of the urine, the milky haze, acid reaction and high specific gravity, together with the chronic persistence of the colon bacilli, when they once appear in the urine. He also notes that the urine never decomposes with an ammoniacal odor. The *urine is always acid.*

Briscoe makes the urine alkaline, gives frequent mercurial purges, and administers autogenous vaccination, the initial dose being 50,000,000 of the dead organisms every seven to eight days, with effective results.

**Wound Infection with the Colon Bacillus.**—The writer has seen a number of cases of wound infection with the colon bacillus, notably several cases after operation for appendicitis. E. A. Babler[1] reports a case following gunshot wound of the liver and colon, and an interesting case of subphrenic abscess with destruction of part of the diaphragm and marked necrosis of the tissues and skin. The condition is depicted in Fig. 236.

Finally, the author reports a case of *general infection* with colon bacilli, in which the organisms were found both in the *urine* and in the *sputum.* This patient ultimately recovered. The case was of such severity and suffered from so many complications that it is described at length.

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[1] Jour. Amer. Med. Assoc., Oct. 29, 1910.

H. G., broker, aged forty-four, operated on at the New York hospital by Frank Hartley, for right inguinal hernia. The subsequent complications were as follows, all due to infection with the colon bacillus: Double pyelitis, cystitis, double pneumonia, purulent bronchitis, two attacks of acute colitis, and a myocarditis.

On March 1, 1910, the patient entered the New York hospital. Previous to admission heart, lungs, and urine were found to be normal. These findings were confirmed at the hospital before operation. His weight was 165 pounds, height 5 feet 11½ inches, was in perfect physical condition, smokes a pipe several times a day, and for two years has not indulged in alcohol except an occasional Scotch-with-soda when dining out. Urine has been normal for two years before operation. Previous to this time a few casts and a trace of albumin were found for a brief period of several weeks. Alcohol was shut off and diet institited, and urine rapidly returned to normal, in which condition it remained. The patient, though very active as to exercise, has always had a tendency to constipation. He has secured bowel action by olive oil internally and other simple remedies.

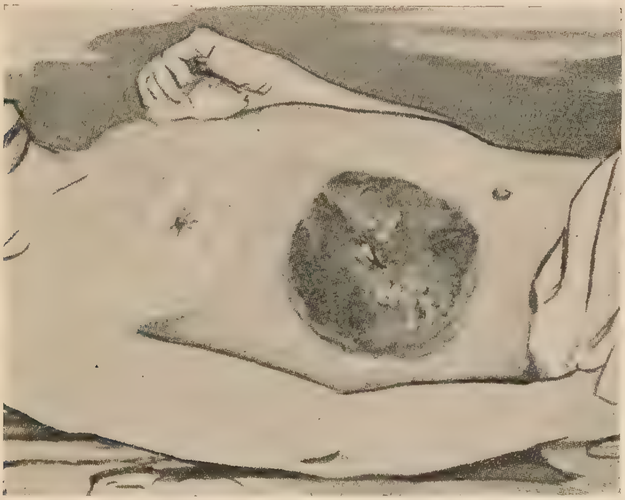

Fig. 236.—Subphrenic abscess (colon bacillus infection) (Babler).

March 2d. Operation for a rather small reducible hernia.

Catheterization was required the afternoon of the day of operation. On the following day there was some irritation of the bladder, which was washed out once, and on the day after this pain in left kidney first began. Urine showed pus—diagnosis, left pyelitis—no examination for colon bacilli was made at this time. Urotropin, 10 grains t. i. d., was given, and an ice-bag was applied to kidney.

The tenderness disappeared within three or four days, and then an attack began in the right kidney, but of not so great severity. The pain in the right kidney rapidly disappeared, but pus persisted in the urine and also a slight temperature. On Friday, March 11th, the patient complained of pains on the right side of the chest, and on March 12th (Saturday) Hartley found a slight dry pleurisy, increasing up to March 13th. On March 14th the writer was requested to examine the patient, and on that date found a small pneumonic area at the right base. The operating surgeon thereupon requested me to take charge of the case. March 16th there was a sudden attack of heart failure, accompanied by a fall in temperature. The patient was nearly pulseless in spite of heroic stimulation and oxygen. Hartley and S. Lambert (the consultants) believed he would die at any moment. Digitalone, 15 drops, and sodium caffein salicylate, 2 grains, were administered every four hours; also strychnin sulphate, $\frac{1}{80}$ grain, every four hours—one given at 2, 6, 10, etc., the other at 4, 8, 12, etc.—so that stimulants were administered every two hours. In addition, the writer had given hourly a hypodermic, 5 grains of camphor in olive oil, for sixteen hours, in all, 80 grains during this period.

The patient's chest was kept thoroughly oiled to prevent blistering, and boiling hot stupes were applied every three minutes over the heart and thorax continuously for seven and one-half hours; then an intermission of one-half hour, then renewal for four hours continuously, and so on. In all, during the twenty-four hours, hot stupes were applied for twenty hours.

As there was some distention, the head of the bed was blocked up to prevent pressure (gas) on the thorax, and the stupes were carried down over the abdomen. The writer believes the application of continuous *extreme heat* and the *camphor injections* were the chief factors in saving the life of the patient in the acute heart failure attack. He was given 40 to 50 grains of camphor daily for some days by hypodermic, in addition to the other stimulants noted. For several days no food was administered, only hot water.

On March 19th some increase in temperature occurred, showing further involvement of the lung at the right base; the temperature at no time was very high, and after two to three days gradually defervesced.

At midnight, March 24th, the patient was suddenly taken with an *extremely severe chill*, lasting about half an hour; the respirations rapidly increased, and when the writer saw him at 1 A. M., March 25th, they averaged 65 to 70 per minute, the heart action was rapid, and the patient was suffering for lack of oxygen. Continuous hot stupes, oxygen, camphor and strychnin, in about an hour gradually relieved the condition, the respirations gradually diminishing in frequency. The temperature shot up to 104° F., then dropped to normal, and then gradually rose to 101° to 101.5° F. Hartley and

S. Lambert saw the case at once, and suspecting the possibility of complicating empyema, Hartley put in aspirating needles in several locations, with negative results.

As there was no apparent cause for this condition, and as the pyelitis was still in evidence, urotropin, 30 grains a day, had been resumed after the acute stage of pneumonia had passed. The writer having had a number of cases of colon bacillus infection with the same type of chill and temperature noted in this case, requested an examination of the urine for this organism, and on suspicion increased the urotropin to 10 grains every three hours, adding sodium benzoate, an equal amount, for the next twelve hours.

In the morning a large number of Gram-negative bacilli were reported under the microscope. The urotropin, 10 grains, and sodium benzoate, 10 grains, were continued every three hours, both day and night, a total of 80 grains each per day, and later pure cultures of colon bacilli were reported in the urine. The temperature gradually defervesced to about 99.5° F., where it remained. Resolution of the right lung was very slow, there was little expectoration, but abundant colon bacilli were found in the sputum. The following events then occurred:

In early May a sharp attack of acute colitis, no blood or occult blood could be found; intestinal irrigations with acetozone (1 : 1000) were given; lactic acid bacilli tablets begun and kept up thereafter.

Early in May a second pneumonia at the left base developed, very slight with mild symptoms; temperature slight and with no cardiac disturbance; it was slow to resolve; undoubtedly the saturation of the patient with urotropin was contributory to its mildness. Colon bacilli were again found in the sputum.

*Blood-culture Taken at this Time and Found Sterile.*—In the middle of May a sudden attack of acute purulent bronchitis occurred. So thick was the pus that pulmonary abscess was feared. Culture showed enormous numbers of colon bacilli and some streptococci. As there was still some expectoration from the pneumonia, the author believes the bronchitis was probably thus directly produced. Beyond the initial few hours there was no especial discomfort except from the cough. Autogenous vaccines were then begun—1,000,000,000 to 1 cubic centimeter. One minim was first administered by hypodermic, and this continued daily for several days, and then gradually increased by ½-minim doses up to the maximum, 5 minims. The bronchitis improved, but there was no disappearance of the bacilli from the urine, though the vaccines were given daily by advice of the pathologist. At the end of May, however, the general condition of the patient had so improved, and the temperature being generally normal the greater part of the day with only a slight evening rise, that he was removed to his home. At the end of three weeks the administration of vaccines caused a marked reaction, so they were stopped.

In June an exacerbation of pyelitis occurred and a second attack of acute colitis, a very suggestive coincidence. Again the stool was examined for pus and occult blood, as ulcer was suspected by the writer, but the results were negative. The author is, however, fully convinced that the colon bacillus infection was autogenous from the gut. A thorough investigation was made at the time of the catheterization, and no flaw in the technic could be detected. The subsequent history of the case proved the theory auto-infective process to be correct.

In June autogenous vaccines were again administered, 200,000,000, and about a week later 300,000,000. Considerable reaction followed these injections, which rather alarmed the patient. They were discontinued and reliance placed on the urotropin and sodium benzoate.

In early July the patient went to Williamstown, at this time there being only a slight evening temperature, 99° to 99.5° F., bacilluria persisting, with few pus cells. There was still a slight evidence of unresolved pneumonia at the left base, but no cough. The patient suffered some shortness of breath on exertion, and the heart action was somewhat rapid, a myocarditis of mild degree, due chiefly to the toxemia in the author's opinion. In early July the writer joined the patient, and twice each week the urine was sent to New York and examined for colon bacilli.

On August 7th the colon bacilli disappeared, there being only a few pus cells found thereafter. The urine remained free from colon bacilli until October. On the first of this month the patient returned to the Stock Exchange and, against advice, persisted in a full day's work. On about the fourth day, he had slight chilly feelings, slight rise of temperature, and a reappearance of the colon bacilli in the urine, though not in great numbers.

These gradually diminished under dosage of urotropin, 50 to 60 grains daily, and there was slight evening temperature only every day or two, to 99° to 99.5° F.

William H. Thomson, on examining the case, agreed with the writer that further physical rest was necessary. He also held that the infection undoubtedly arose from the intestinal tract through some undiscoverable lesion in the intestinal canal. The patient was sent to Europe to a warm climate, where he remained until May 1, 1911. He continued urotropin, about 40 grains daily, until the disappearance of the colon bacilli; thereafter urotropin, 15 grains daily, until his return.

The patient, December 24, 1910, wrote me that his temperature remains normal, and only a few colon bacilli persist in the urine. In January, 1911, the colon bacilli entirely disappeared from the urine. The subsequent course of this case is of considerable interest. From January, 1911, to July, of the same year, no colon bacilli were found in the urine. In May the patient returned to the Stock Exchange. In July an attack of acute tonsillitis occurred, with immediate reappearance of colon bacilli in the urine. The writer was absent from the city, and the physician in attendance did not examine the throat discharge. It would have proved interesting to note whether an exterior reinfection could have occurred through the tonsils. From January to May 1, 1911, the patient had continued urotropin, 15 grains daily in divided doses, as a prophylactic, and thereafter took occasional doses up to the time of recurrence. The writer advised a return to 60 grains of urotropin a day at this time. The patient rapidly recovered from the tonsillitis, felt well, and in about a week resumed business, but the bacilluria was still present. About October 15, 1911, an occasional colon bacillus was found in the urine, though the patient was apparently well. Urotropin, 15 grains daily, was still continued. The case is particularly interesting from the fact of recovery from apparently what was believed to be a fatal infection and from the tendency to recurrence. The tendency to chronicity of this infection is undoubtedly difficult to combat. The patient before October 1st had regained his loss of weight, which was 35 pounds. Urotropin, 80 grains, with sodium benzoate, 80 grains, were given daily from March 25th to June 1st. From June 1st to July 9th, urotropin averaged 60 grains daily. Up to May 1st no irritation resulted from the treatment. Sudden changes in the urinary reaction then occurred, and there was considerable difficulty in controlling the irritation. It would be extremely acid at one time and very alkaline later, both conditions causing irritation. The writer finally secured the best results keeping *the urine neutral* or faintly acid, and stopping the benzoate when a sudden excessive irritation with acidity occurred, and then giving an alkali (citrate of potash) with the urotropin. On the other hand, when the urine was alkaline with irritation from too much alkali, sodium benzoate was given with the urotropin and the potash stopped. The urine was carefully watched for the presence of casts and cylindroids, but no evidence of renal irritation from the urotropin could at any time be discovered. From July 7th to August 7th, urotropin, 50 to 60 grains, was administered daily.

From August 7th to October 4th, urotropin was reduced to 40, 30, and, finally, 20 grains daily, the latter as a prophylactic. In spite of this the colon bacilli reappeared in October. It was interesting to note that the 30 grains daily of urotropin given for the initial pyelitis did not prevent the nearly fatal colon bacillus pneumonia, and that the large doses (80 grains daily) did undoubtedly prevent severe symptoms in the subsequent second pneumonia and purulent bronchitis. The autogenous vaccination evidently was of some value to relieve the symptoms of the bronchitis, but the disappearance of the *bacilli only occurred* following the persistent use of urotropin. Hugh Cabot[1] has shown that the use of the vaccines is followed by *improve-*

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[1] Amer. Assoc. of Genito-Urinary Surgeons, Eighth Congress of American Physicians and Surgeons, 1910.

*ment in the clinical symptoms in one-half the cases*, but that they *have but little effect on the bacteriuria.*

There was considerable reaction toward the end of the first period of autogenous vaccination, and marked after the second.

Finally, the author wishes to call to the reader's attention that, in addition to the marked indicanuria in intestinal obstruction, colon bacilli infection may also occur. The possibility that gastric hemorrhage, as a complication of acute appendicitis, may be the result of colon infection carried to the stomach, is worthy of investigation, especially in view of Türck's experiments.

*Diagnosis.*—Urine should be examined for Gram-negative bacilli (colon bacilli), and then cultures should be grown; wound infection should be cultured, and also the discharge from tumors or infected wounds.

*Urine Reaction.*—With *colon infection* and tubercle infection the urine is *always acid*. This is an aid to diagnosis. Other types of pus and urine, with pyelitis or cystitis, give an alkaline reaction in the urine.

*Treatment of Infection by the Colon Bacillus.*—This may be summarized as follows:

(1) Urotropin and sodium benzoate, each 10 grains, every three hours by mouth; by rectum, if there is vomiting or coma. After prolonged use, if excessive acidity with irritation occur, omit the benzoate and give, with the urotropin, potassium citrate; thus quickly regulate the urine so as to be neutral or faintly acid. Thomson has shown that the sodium benzoate seems to augment the effect of the urotropin and lessens the chance of bladder disturbance. As the condition improves the urotropin and sodium benzoate may be gradually reduced.

(2) Autogenous vaccination, especially if the infection is chronic or does not respond to treatment, or there is additional infection elsewhere, 50,000,000 increasing to 300,000,000 to 500,000,000 gradually, first given every two or three days; larger doses at longer intervals, four to seven days. Some advise 200,000,000, increasing to 1,000,000,000.

(3) Lactic acid bacilli tablets should be given t. i. d.

(4) High enemata—1 : 1000 acetozone, 1 quart every day, and later every other day—are of value.

(5) Sour-milk diet: bacillac, fermillac, matzoon, lactone-buttermilk, and koumiss; later, cereals, etc.; *avoid red meats.*

(6) Bowels should be opened at once by calomel or blue mass and then regulated carefully daily. Calomel or blue mass once a week.

(7) Antibacillus coli serum,[1] 25 c.c. in divided doses in three days' time, has been recommended.

(8) Surgical procedure when indicated.

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[1] Rawls, Med. Rec., Oct. 7, 1911.

## CHAPTER XXIII

# CONSTIPATION AND DIARRHEA

### CONSTIPATION

(*Synonyms.*—Obstipatio Alvi; Constipatio Alvi; Atony of the Bowel; Habitual Constipation.)

HEALTHY persons usually have one bowel movement daily, generally about the same hour. Some normally have two actions a day, while others empty the bowels every other or every second day, and yet are in perfect health.

Constipation is defined as a condition in which the feces are not passed sufficiently often. Another form is that in which defecation occurs daily, but the movements are insufficient in quantity. A stagnation of fecal matter may thus occur. The quantity of feces is somewhat variable, from 100 to 150 grams, the average, even up to 250 grams, being greater after a vegetable diet and less after meats.

Considerable of the evacuation is made up of micro-organisms, of which Herter[1] has estimated the daily number as 126,000,000,000, which explains the fact that patients who eat little may pass considerable material.

Constipation may be acute or chronic. The acute type is due to complete obstruction of the intestinal tract or to dynamic ileus (intestinal paresis).

The chronic type is extremely common. Henry Illoway and Samuel Gant have written excellent works on this subject. My classification is slightly modified from their books.

**Etiology.**—All possible factors must be carefully investigated and corrected.

1. *Diseases of the stomach*, such as hyperchlorhydria, ulcer, cancer, dilatation, simple atony, catarrhal conditions, and achylia gastrica, may cause constipation.

2. *Obstruction of the bowel* by tumors of the intestine or tumors pressing on the bowel from some adjacent organ; by *stricture within the intestine;* by external stricture, as by peritonitic adhesions; by chronic intussusception.

Lane describes a type of chronic obstruction with dilatation of the small intestine, including the duodenum, due to partial obstruction of the termination of the ileum by a mesentery, or band developed in the mesentery, by an anchored appendix or by pressure of the ileum on the pelvic brim. Mayo[1] also refers to cases of chronic intestinal stasis due to kinks and adhesions of the terminal ileum (the Lane kink) frequently associated with chronic appendicitis.

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[1] Bacterial Infections of the Digestive Tract.

3. *Catarrh of the small intestine alone;* in some cases catarrh of the large intestine; mucous colic; atrophy after catarrh; ulcers of the small intestine are occasionally attended by constipation; *dysenteric ulcers at times produce constipation, though ulcers of the large intestine usually cause diarrhea.*

4. *Voluntary abstention from stool* on account of the pain it produces, by reason of disease of the rectum, such as from piles, fissure, or ulcer. Increased contraction of the sphincter ani (irritability), at times with resulting hypertrophy, also occurs from irritation and interferes with bowel action. Hypertrophied levatores ani muscles which may be felt as thick rigid bands about 2 inches above the anus may clump the rectum and interfere with bowel movements.

5. *Obstruction to the entrance of bile into the intestine* or deficiency of bile.

6. *Diseases of the heart, lungs, liver, and kidneys.* Intestinal hyperemia and congestion of the portal system are factors in these cases in retarding peristalsis, as in cirrhosis of the liver.

7. *Disease of the pancreas.*

8. *Diabetes*, anemia, and chlorosis.

9. In *many diseases of the brain, spinal cord, and of the nervous system* constipation is present.

Among such are chronic insanity, diphtheritic paralysis, tabes, brain tumors, cerebrospinal meningitis, hemorrhage of the brain, chronic hydrocephalus, myelitis, neuroses, and psychoses.

10. *Acute febrile conditions are usually accompanied by constipation*, as pneumonia, etc. This generally excludes those with special intestinal lesions. We must remember that constipation occurs in some cases of typhoid, and even of dysentery.

11. *Chronic constipation from foreign bodies.* They may be lodged in the bowel or beneath the mucosa and either obstruct the rectum or cause sphincteric contraction.

12. *Malformations*, such as abnormally developed colon; undue size or length of the sigmoid flexure; diverticula of the large intestine; a diaphragm partially closing the large intestine.

13. *Defective development or essential primary atrophy of the colon.*

14. *Enteroptosis;* angulations of the sigmoid flexure due to adhesions; prolapse of sigmoid into rectum from long mesentery—in effect, slight intussusception; cecum mobile (movable cecum).

15. *Atrophy of the intestinal musculature* following catarrh or fatty degeneration, as in consumption or in alcoholics.

16. *Hypertrophy of Houston's*[2] *valves*, or hypertrophied O'Bierne's sphincter, which is located at the rectosigmoidal juncture, may be

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[1] Surgery, Gynecology, and Obstetrics, March, 1911.

[2] Gant, N. Y. Med. Jour., April 15, 1911.

causes. Coccygeal deviation with projection inward of the bone may be a factor.

17. *Loss of power in the abdominal muscles* may be a factor in some cases, as in the emaciated, with multiparous women with diastasis of the recti, etc.

18. Distention of the duodenum secondary to compression or obstruction of the third part of the duodenum by reason of a strain exerted on it by the mesentery of the small intestine or by the pull on it by the jejunum. Some of these cases have associated a duodenal ulcer, and Lane[1] describes a case in which there was torsion at the duodenojejunal junction, the patient being a sufferer from chronic intestinal stasis, a stenosis of the pylorus being suspected.

19. Chronic constipation *from impaired physiologic function.* This type is due to disturbance of the motor function of the intestines, and is strictly classified under *motor neuroses*, under which we have:

(*a*) Constipation due to retarded intestinal peristalsis (atony or relaxation of the bowel).

(*b*) Spastic constipation, perverted action, or enterospasm. The constipation is due to a spasmodic contraction of a portion of the intestine.

(*c*) Spasm of the sphincter is included under this type.

*Constipation Due to Disturbances of the Motor Function.*—Habitual constipation due to impairment of the physiologic function (*i. e.*, caused by motor disturbances) constitutes an important class of cases. It should be strictly classified under *motor neuroses.*

There are the two types mentioned above: the atonic and the spastic forms of constipation.

*Atonic Constipation.—Constipation due to atony* (literally, relaxation) *of the bowels* constitutes the majority of cases.

*Predisposing Causes.*—The character and quantity of food and the amount of physical exercise influence bowel evacuation. Boas has laid stress upon the importance of the type of nourishment and its influence in the production of constipation (alimentary constipation).

Albuminous diet consisting of meat and eggs leaves little residue, and eventually tends to constipate; while with vegetable diet there is more residue and the fermentation products excite peristalsis. A patient may diet by avoiding vegetables, butter, and fat, and thus constipation result which may become chronic.

*Repeated neglect of the call of nature is a frequent cause*, such as in the case of young girls and children while at school, or among women at social gatherings, etc.

Interruption of the habit of regularity, as on a railroad journey, or an attack of diarrhea with subsequent constipation, or of acute fever, may lead to habitual constipation. The same is true of mental conditions, such as depressing emotions. Prolonged exercise, such as long marches, has produced chronic constipation.

[1] Surgery, Gynecology, and Obstetrics, March, 1911.

Constipation is more frequently found among the wealthy than among the working class. The mode of living, sedentary life, etc., have a bearing. Worry and mental strain have an influence, and it occurs among neurasthenics and hypochondriacs.

Habitual constipation is found in those engaged in active exercise and who are of strong constitution. In such persons the cause of this perversion of motor function is unknown, but it seems inherent to the patients that the bowels respond slowly to stimuli. In some cases there seems to be hereditary or congenital diminution of intestinal peristalsis (constipation).

Riegel observed cases in which at autopsy the musculature of the large intestine was abnormally thin and was probably congenital. In some of these cases the skeletal muscles were strong. Nothnagel described similar cases in which the general muscular development was poor. These rare conditions cannot be recognized *intra vitam.*

Enteroptosis is given as a cause of chronic constipation on account of the intestinal angulation which occurs. This is true in some cases. The general atony[1] with enteroptosis is the chief factor. The musculature of the stomach, intestines, and also the abdominal muscles are relaxed, which have a bearing on the production of constipation. The abuse of cathartics, and repeated distention of the bowel from large enemata, may produce constipation.

*Spastic Constipation.*—Spasmodic constipation of the bowels, or enterospasm, is produced by a perversion of the motor function of the intestines, taking the form of a spasmodic contraction of a portion of the intestines, which may involve both the circular and longitudinal muscles.

This spastic condition may be of variable duration and involve different segments of the intestines. Fleiner holds that the contracted portion retains its contents, while others believe that it is nearly occluded by the spasm, thus creating an obstacle to the passage of the intestinal contents.

Diffuse enteropasm involving the small intestine occurs in spinal meningitis, in diseases of the pons and medulla, and in chronic lead-poisoning. In these cases the abdomen is retracted like a trough.

Local or circumscribed enterospasm is more frequent, and it generally affects a portion of the large intestine. The abdomen shows no abnormality on inspection.

Spastic constipation is more frequently met with in nervous persons, neurasthenics, the hysteric, and in those debilitated by long-continued disease. The constipation is quite obstinate, lasting for several days. The evacuation is somewhat painful and consists of small balls (goat feces) or pencil-shaped fecal material. There are spasmodic pains in the left lower abdomen or in the umbilical region which are relieved by the passages.

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[1] Rose and Kemp, Atonia Gastrica.

*Spasm of the Sphincter (Proctospasmus).*—This condition really belongs to spastic constipation. There is painful spasmodic contraction of the sphincter. I have seen cases, however, in which pain was not marked, but spasm occurred when defecation was attempted; in effect, spasmodic stricture was present. Such cases may be a direct factor in the production of constipation.

Many cases of sphincteric spasm are secondary to fissure or ulcer of the rectum, or are reflex when there are inflammatory conditions in the neighboring organs, such as the uterus, bladder, etc. Some cases occur as a primary nervous affection and are chiefly met with in those with a nervous taint or disease of the spinal cord.

Rectal examination is often extremely painful. In some cases there is simply reflex spasm on examination, and the sphincter is found to be extremely tight and evidently hypertrophied from frequent contraction.

It is important to consider the possibility of the last type in connection with chronic constipation.

**Symptoms.**—In many cases constipation causes no subjective symptoms. Some have a stool every second or third day or even once a week. There is a classical case *who had an annual movement.* Nevertheless, chronic constipation should not be made light of.

Some patients on the appearance of constipation immediately suffer from subjective symptoms, which may become at times quite severe. Among milder symptoms are sensations of fulness, tension, and discomfort in the abdomen; at times they are referred to the stomach; loss of appetite, occasionally belching, nausea, pyrosis, and a disagreeable taste in the mouth. Coated tongue and headache are often present. Colicky pains and distention may occur.

**Intestinal Sapremia.**—In cases of marked constipation severe symptoms may occur, such as headache, apathy, a dry furred tongue, anorexia, prostration, abdominal pain, sustained elevation of temperature, palpable spleen, and even an eruption, so that typhoid may be suspected. The eruption is profuse and blotchy, macular and erythematous, and not likely to be mistaken for typhoid, roseola, or Brill's disease. Physical examination will determine fecal accumulation.

These symptoms disappear after thorough evacuation of the bowels. In cases of doubt, such as intestinal sapremia of a duration which is not definitely obtainable, it may be advisable to make the laboratory tests to differentiate from typhoid fever.

On inspection and palpation considerable fecal accumulation can be found in the intestines, especially in the colon. Some patients suffer from severe headache, dizziness, sleeplessness, despondency, palpitation, tachycardia, or irregular pulse. Such symptoms I believe due to auto-intoxication from the intestinal tract. *Motor insufficiency, as in constipation, favors stagnation and putrefactive changes.*

Duprey[1] reports extreme cases in which the patients became unconscious, but recovered after free bowel evacuation. One death also occurred.

In most of my patients suffering from chronic constipation, with the symptoms just described, especially with nervous manifestations, marked indicanuria was present.

Herter[2] states that most children and many adults may fail to develop indicanuria with constipation, yet there are others in whom it is marked, and that a *satisfactory explanation is not possible.*

In my report of 13 cases of dementia paralytica before the American Medicopsychologic Association[3] marked constipation was noted in all the cases, and all suffered with considerable temperature. Rectal irrigation lowered the temperature in 3 cases, and under general treatment for the gastro-intestinal tract it was lowered in 8 more. Convulsions diminished in 5 cases. The bowels were freely opened.

Bouchard's theory, that no intoxication from the intestinal tract can take place when the feces are solid, I believe untenable. Dunin has shown that constipation may be the result of nervous conditions. There are many in which no nervous factor can be discovered, and others in whom constipation and intestinal auto-intoxication are factors in the production of nervous symptoms.

**Termination.**—The bowels may act spontaneously, hard masses of fecal matter being passed, covered with a thin layer of mucus. Feces are often passed in small balls or in rod shape. With the atonic type of constipation relief is usually felt after defecation, while with the spastic type movement is accomplished with great effort, and there is a feeling as if there were still material in the rectum.

In some cases the constipation terminates in an attack of diarrhea due to hyperemia and the secretion of fluid from irritation of the mucosa by the hardened feces. In others, purgatives or enemata may be required, or removal of scybalæ from the rectum by the fingers.

Slight catarrh may occur or, rarely, stercoral ulcers, local peritonitis, or even perforation and general peritonitis. Constipation has been a factor in the production of typhlitis, diverticulitis, catarrh of the cecum with secondary catarrhal appendicitis, volvulus, and subacute or acute intestinal obstruction.

**Fecal Colic.**—When large masses of fecal matter accumulate colic may occur. The patient is seized with violent colicky pains which may cause a fainting spell. The abdomen is distended and tender. Passage of flatus brings temporary relief. The symptoms do not disappear until thorough evacuation of the fecal accumulation takes place. Fecal colic may occur, with daily evacuation of the bowels. The detection of fecal accumulation by palpation is of chief importance.

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[1] The Lancet, 1902, p. 1832.

[2] Bacterial Infections of the Digestive Tract, p. 263.

[3] Proceedings of the Sixty-first Annual Meeting, April, 1903. Some observations on the Relations of the Gastro-intestinal Tract to Nervous and Mental Diseases.

In obstinate cases of constipation cathartics may fail to produce movements and the patient go into marked collapse and vomit profusely, with symptoms resembling intestinal obstruction. Rectal irrigations or oil injections may relieve the condition.

Rarely, in the insane, old, or weak, total paralysis of the colon may take place and the patient die with the symptoms of obstruction. In cases of fecal accumulation it is always safer to employ injections and irrigations before resorting to active catharsis.

**Fecal Tumors.**—They occur most frequently in the cecum, sigmoid flexure, rectum, and hepatic and splenic flexures. They may cause dislocation of the transverse colon, and the mass be felt a short distance above the symphysis. In most cases the *tumor is not of very firm consistency, is movable, and pits on pressure.* On the other hand, it may be nodular, hard, or angular.

In some cases the bowels may move every day, there evidently being a free central passage.

*Gersuny's Adhesion Symptom.*—If the abdominal wall over the tumor is gradually depressed with the finger-tips, the pressure gradually diminished, and the fingers slowly withdrawn, one can feel the mucous coat of the intestines loosening itself from the feces forming the tumor (*i. e.*, the wall of the bowel separates from the tumor when palpating pressure is relaxed).

If under intestinal irrigations, etc., the tumor diminishes in size, it is evidently fecal. Anesthesia may rarely be required for examination.

Some of these fecal tumors have developed into large size—over 4 pounds or more—and after the colon is dilated, stercoral ulcers, local or general peritonitis, or intestinal obstruction from kinking, compression, torsion, or internal occlusion may result.

Hemorrhoids are a complication. They are described in a special section.

I have referred to *various nervous symptoms that are dependent on constipation.*

Leube describes intestinal vertigo, which he believes reflex and due to pressure on the hemorrhoidal plexuses of the sympathetic, since the palpating finger in the rectum also produced it.

Senator imputed dizziness and vertigo to absorption of poisonous gases, such as sulphuretted hydrogen; while Nothnagel assumes that nervous symptoms are due to absorption of ptomains, thus causing auto-intoxication.

Auto-intoxication I believe to be the correct explanation. In persons of a neuropathic disposition auto-intoxication from chronic constipation is undoubtedly a contributory factor in the production of nervous disease, especially melancholia.

**Fecal Fever.**—This is generally due to some complication, such as inflammation, local peritonitis, or stercoral ulcer. With infants and young children fever may be caused by fecal accumulation. I have referred to the cases of dementia paralytica with temperature, with disappearance of the latter after bowel action.

Clark holds that chlorosis is the result of toxins absorbed from coprostasis. Hyperchlorhydria is generally present and this would have an influence on the bowels.

Though diarrhea with marked multiplication of the Bacillus aërogenes capsulatus often occurs in common with severe primary anemia, and in some instances the cases present the blood-picture and clinical characteristics of pernicious anemia, yet Herter shows that advanced infection with the gas bacillus can occur[1] without any diarrhea, in fact, with *obstinate constipation.* Stasis is favorable to putrefaction. The possibility, therefore, of stasis (constipation) favoring in some cases the development of poisons having a hemolytic action on the blood should be considered.

**Diagnosis.**—The irregularity of the movements and their character, occurring in small balls, pencil shape, or in small fragments, and abdominal palpation, disclosing fecal masses, are diagnostic. One must remember that daily incomplete evacuations may occur.

Fecal masses are found most frequently in the caput coli, sigmoid, and the rectum. It is always well to examine the latter. *All possible causes of constipation must be considered.* If it is suspected to be due to anomalies of the functions of the stomach, gastric analysis must be performed and treatment directed toward the gastric condition.

Diseases of the heart, lung, liver, and kidneys must be treated if present. Rectal examination may disclose hypertrophy of Houston's valves; in such event, their division by the application of Gant's valve clamps is indicated.

Prolapse of the sigmoid may be determined by inspection with the sigmoidoscope. Gant's suspensory operation is then indicated. Splanchnoptosis if present must be treated and Rose's belt applied.

If none of these causes can be determined, the constipation must be purely functional (habitual), either of the atonic or spastic type.

With atony of the bowels there are slight bloating, evacuation of hard fecal matter, often in balls covered with a thin layer of mucus, at times dizziness and nervous symptoms. *Severe pains are rare.*

With enterospasm there are uneasiness and pain at the time of evacuation or just preceding it, and at times spells of faintness. The fecal matter is not so hard and is evacuated after considerable straining in narrow, tape-like or pencil-shaped pieces. There is no feeling of complete relief after evacuation, but as if more were present. The abdomen may at times be sunken and retracted and the intestinal coils can sometimes be palpated.

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[1] Bacterial Infections of the Digestive Tract, p. 207.

**Prognosis.**—This is favorable in the majority of cases as regards life, though occasionally incurable lesions, such as diverticulitis, ulceration, or even peritonitis and death may result. These complications are comparatively rare. The prognosis as to cure depends on the cause of the constipation. In the functional cases of habitual constipation cures result, but many cases require more or less care for the balance of their lives.

**Treatment.**—*General Methods.*—The cause of the constipation should be treated. Persons who for years have habitually had a movement every second or third day and are in good health should be let alone.

*Prophylaxis.*—Never place a patient on a restricted diet for too long a period, excluding vegetables, fruits, starchy foods, and fats which would dispose to constipation. Abuse of cathartics should be avoided.

A hygienic method of living, proper out-of-door life and exercise, diminution of strain and worry are necessary. As few purgatives as possible should be employed.

*Training of the Patient.*—One should allay the patient's anxiety. He should be told not to worry if the bowels fail to act for a day or two. Meanwhile, rational methods should be undertaken by the physician to produce the desired result. Trousseau first advocated teaching regularity. The patient should be taught to endeavor to have an evacuation at a regular time every day, preferably half an hour after breakfast. He should go to the closet and try to have a passage, but should not exert himself for over five minutes. It is an excellent procedure to aid the return to the habit by insertion of a small gluten or glycerin suppository or, preferably, the injection of 1 to 2 ounces (30.0–60.0) of olive oil with a soft-rubber hand syringe. This is better than waiting twenty-four hours, as the desire is often thus stimulated. Regular habits could thus often be cultivated and the small injection then stopped.

*Diet.*—The main object is the ingestion of foods which increase intestinal peristalsis and the avoidance of constipating material.

A glass of cold water, or, in some cases, of hot water, should be taken on rising. Water should be taken on the fasting stomach and a moderate amount—8 to 10 ounces (250–310 c.c.) at least—of fluid at meals. Total abstinence from liquid at meals tends to constipate. The following are of value: buttermilk, cider, sour milks, fermented milk, such as lactone-milk, koumiss, matzoon, bacillac, carbonated waters; raw fruits, such as grapes, oranges, grape-fruit, apples, figs, prunes, cranberries, pears, peaches, plums, mandarins, gooseberries, currents, strawberries, raspberries, blackberries; cooked fruits, jams, treacle, honey, lemonade; vegetables rich in cellulose, cabbage, cucumbers, spinach, green peas, Brussels sprouts, cauliflower, green salads, turnip tops, Spanish onions, carrots, asparagus; syrup, sugar; salmon, sardines, herring; rye bread, Graham bread, brown bread, pumper-

nickel; fatty and highly seasoned foods, plenty of butter, cream, and fat cheese. Excess of potatoes or rice constipate. Oatmeal is often of value. Milk is constipating with some and purgative with others. Whortleberries are constipating. Red wines, tea, chocolate, and cocoa should be avoided. Beer and champagne are recommended by some. Chicken and red meats are given in moderation.

We may say the diet should be mixed, with a preponderance of vegetable food. Some patients, of course, have idiosyncrasies to certain foods, and one would not give a patient, with a delicate stomach, cabbage, cider, and brown bread. If the intestines are already overburdened with too much ballast and excess of cellulose has been given, such articles must be restricted.

The internal administration of *olive oil*, 1 to 2 ounces (30.0–60.0) or more, has often an excellent effect; to be given once or twice daily.

If fermentative or putrefactive processes are present, they must receive attention.

In very obese patients one would not give excessively fattening food. Often a few prunes with the morning glass of water and fruit for breakfast are serviceable. The administration of a raw apple thoroughly masticated at 9 P. M., and an hour later a large glass of Vichy, I have often found valuable, a morning movement resulting.

As a sample diet one may give as follows:

*Breakfast.*—Oatmeal with cream or porridge with syrup, fat bacon, rye bread with plenty of butter, marmalade or honey, coffee with cream, water 8 ounces (250 c.c.); grape-fruit or oranges.

*Lunch.*—Fish, potatoes (very little), green vegetables, salad with plenty of oil, stewed apples or figs, wholemeal bread, butter, fruit, water, or lemonade.

*Dinner.*—Tomato or some vegetable soup, meat, spinach, string-beans, asparagus, salad with oil, dry toast or biscuit with plenty of butter, apple charlotte, stewed pears or prunes, cheese, uncooked fruits, water or lemonade.

*Physical Methods.*—These are useful to strengthen the bowel and promote better action or to directly stimulate peristalsis.

*Massage.*—This is of use in the atonic cases, but not as much so in spastic constipation. It should, preferably, be administered by an expert and the treatment carried out for many months.

Abdominal massage should be carried out in the course of the colon by short tapping motions (vibratory), or by kneading and rubbing. It is preferable to massage from the caput coli to the sigmoid. The small intestine should also be manipulated as well as the abdominal muscles.

Illoway,[1] recommends massage for five to fifteen minutes in adults and three to five minutes for children, at least every other day, for a period of six weeks, and then, if there is improvement, at longer intervals, but for a long period of time. It should be given preferably early in the morning in the fasting condition.

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[1] Constipation in Adults and Children.

*Automassage.*—The patient sitting upright with the right hand should stroke the abdomen from the caput coli to the hepatic flexure, and then along the transverse colon. With the left hand he can then massage down the descending colon. Circular stroking movements should then be made over the median abdominal region. I often have the patient follow this out while endeavoring to have the morning defecation. Séances should last about five to ten minutes.

*Cannon-ball Massage.*—A 3- to 5-pound cannon-ball rolled over the abdomen in the course of the colon and small intestine spirally is of value. The patient can employ this in the dorsal position.

The hollow *wooden* cannon-ball with a screw-cap, arranged so that shot can be placed therein, and thus different weights employed with the same ball, is an excellent instrument (Fig. 237). It is arranged with a handle and frame so that it can be more easily manipulated (Fig. 238). One should begin with a weight of 2 pounds and increase it gradually to 5 pounds.

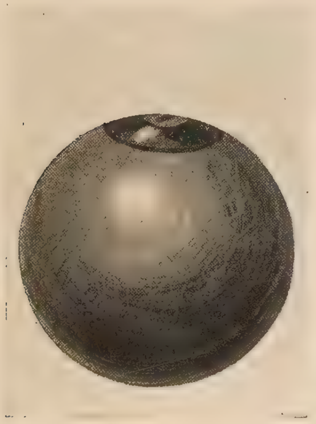

Fig. 237.—Cannon-ball with screw cap.

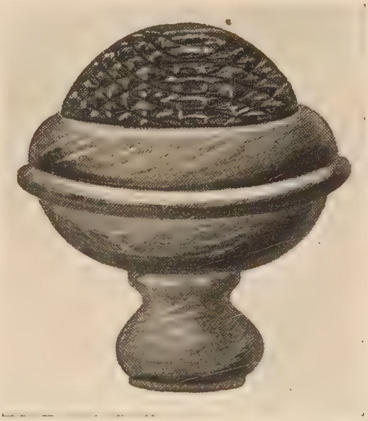

Fig. 238.—Cannon-ball with handle.

*Vibratory Massage.*—This is of great value. Special vibration should be given over the sigmoid flexure. The Vedee vibrator can be used alone or with electricity, or the Eureka or any good electric vibrator can be attached to the street current. Séances should last about ten to fifteen minutes in the course of the colon and over the small intestine.

*Hot and Cold Massage Electric Roller.*—This instrument has been described and can be employed for the atonic cases.

*Gymnastic Exercises.*—Exercises which bring the abdominal muscles into play are of value, such as gymnastics on the horizontal bar, horseback riding, hill climbing, skating, rowing, bicycling, golf, tennis, boxing, fencing. Overexertion and too abundant sweating should be avoided. Flexion and extension of the body and lateral rotation while in the sitting posture; bending downward and then upward in the standing position with the knees held stiff; Swedish movements and treatment by the Zander methods are all of value.

*Electricity.*—Percutaneous electricity, especially faradization over the abdomen, is useful as an adjunct. The intrarectal method (one electrode in the rectum and the other over the abdomen) is recommended, especially galvanization.

The author's method with *recurrent electric irrigation* is practical. With a glass Y attachment and two fountain syringes, alternate hot and cold electric douches can be employed for the atonic condition. I have used both the faradic and galvanic current. In obstinate cases of high impaction I have found hot normal saline douches at 120° F. with the faradic current most efficacious, duration fifteen to thirty minutes.

Kussmaul has suggested one electrode in the stomach and the other in the rectum.

Electricity is indicated in the atonic cases. Static electricity is also recommended. Doumer[1] has employed it in the form of sparks, especially in the left iliac fossa.

*Hydrotherapy.*—Frictions, cold douches, the alternating cold and warm fan douches, Scotch douches, short cold sitz-baths (five minutes at 12° C.), the wet binder (Neptune's girdle) applied over night, and the Priessnitz compress are all recommended. Hydrotherapy must needs be conducted at a sanatorium.

For practical purposes the sitz-bath and wet abdominal compress suffice the general practitioner.

*Injections; Enteroclysis.*—Recurrent enteroclysis with hot normal saline solution at 110° to 120° F. for fifteen minutes three times a week, alone or combined with electricity (rectal method) or with the alternating cold douche at 60° F., may prove of service in the very obstinate cases.

Enteroclysis with flaxseed tea has also proved valuable.

The soapsuds and water enema alone—1 pint to 1½ quarts (500–1500 c.c.)—or with live oil, 8 ounces (250 c.c.), or castor oil, 1 to 2 ounces (30.0–60.0), included, or normal salt solution alone, may be required, depending on the conditions. A high enema of olive oil, 8 ounces (250 c.c.), combined with glycerin, 4 ounces (125 c.c.), is often useful.

Often a small enema of normal saline or soapsoads and water of 1 pint (500 c.c.), if given with the patient in the knee-chest position is more efficacious than the larger injections. The water injections may be employed at the same hour daily for a considerable time. The large injections, as recommended by some, overdistend the already atonic intestines. The Sims' position is excellent for the injection.

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[1] Annales d'Electro-Biologie, 1898, p. 722.

Klemperer[1] recommends the injection into the bowel of small quantities of water at bedtime—only $\frac{1}{2}$ pint (250 c.c.)—and the patient is told to retain the fluid. It is soon absorbed and evacuation occurs the following morning. These injections are given every night for three weeks, and then every other night.

Kussmaul and Fleiner employ an injection of sweet oil into the rectum at bedtime, which is to be retained. I believe it advisable to start with a small quantity, only 4 to 6 ounces (125.0–200.0), heated to the temperature of the body and slowly injected through a colon-tube from a fountain syringe. The patient should retain the oil as long as possible (over night if he can). Gradually increase the quantity to 8 ounces to 1 pint (250–500 c.c.), and in obstinate cases nearly to 1 quart (liter).

As a rule, evacuation follows the next morning. I give the injection every night for a week, then every other night for several weeks, then twice a week, and, finally, once a week. The treatment should cover several months. This method is recommended especially for the *spastic type of constipation*, but I have found it of value in other cases.

Olive oil, 1 to 2 ounces (30.0–60.0), by mouth once to three times a day is a valuable adjunct. Cottonseed oil can be *substituted by enema* for olive oil.

*Glycerin Injections.*—Glycerin, 1 to 2 drams (4.0–8.0), dissolved in 3 ounces (95.0) of water and injected into the rectum is of service in some cases, or given as a suppository. It is sometimes irritating.

Flatau[2] inserts or insufflates into the rectum 15 to 45 grains (1.0–3.0) of boric acid powder. Bowel action results one-half to three hours later.

*Orthopedics.*—I have found Rose's belt of value in chronic atonic constipation, even if no ptosis is present. It lends strength to the abdominal muscles and so aids evacuation.

*Medication.*—In many cases of constipation mild laxatives must be employed, sometimes only temporarily. More powerful cathartics are often required. In constipation of the *spastic form* and in the atonic type with fecal impaction, belladonna is of great value. It should be given preferably as the tincture, in large doses, 10 to 15 minims (0.592–0.888 c.c.), and pushed three or four times a day, so that physiologic symptoms are apparent. In constipation due to atony, strychnin, $\frac{1}{50}$ to $\frac{1}{30}$ grain (0.00108–0.0021) t. i. d., or tincture of nux vomica in 5- to 10-drop doses are of value, even if no laxatives are given. They are often combined with a laxative.

Among the milder laxatives are fluidextract of cascara sagrada and the aromatic fluidextract of cascara, of which the dose is 1 to 2 drams (4.0–8.0); extract of cascara, 1 to 5 grains (0.065–0.324); laxophen, a solution of phenolphthalein, 1 to 4 drams (4.0–16.0);

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[1] Therapie der Gegenwart, 1899, p. 48.

[2] Berlin. klin. Wochenschr., 1891, p. 231.

phenolphthalein (purgen),[1] 1 to 5 grains (0.065–0.324) to 15 grains (1.0); phenolax (1 to 3 wafers); phenolphthalein may be given in capsules 1 to 5 grains each at bed time or even t. i. d.; purgatin, 15 to 30 grains (1.0–2.0), which last is contraindicated in renal disease.

Ad. Schmidt[2] claims that the internal administration of agar-agar, cutting up the straws into small fragments and administering as much as 25.0 grams a day, aids in softening the feces and also evacuation. He adds to it 25 per cent. of an aqueous extract of cascara sagrada. Schmidt recommends this combination, which is dispensed as "Regulin," as being of value in chronic constipation. Dose, 1 teaspoonful (4.0) to a tablespoonful (16.0) or more, mixed with stewed apples or mashed potatoes. It can be secured as tablets. Agar-agar may be employed alone. The writer believes regulin to be of considerable value. It can be given t. i. d. if required.

Syrup of tamarinds, 1 dram (4.0), or a sauce of tamarinds; syrup of figs, 1 to 2 drams (4.0–8.0); compound licorice powder, 1 to 4 drams (4.0–16.0). This last gripes some patients. Olive oil by mouth, 1 to 2 ounces (30.0–60.0), several times a day is valuable.

*Aloes* does not lose its effect even when employed for a long time, and painless defecation results. It may be used alone or in combination. If hemorrhoids are present I do not advise it.

Rhubarb is an excellent drug. Pilula aloes, dose, 1 to 2 pills at night. Tincture rhei aromatici, ½ to 1 dram (2.0–4.0). Tincture rhei, 1 to 2 drams (4.0–8.0). The following are of value:

| | | | |
|---|---|---|---|
| ℞. | Tinct. nucis vomicæ | ʒiss | 6 |
| | Fl. ext. cascara | ℥ss | 16 |
| | Pulv. ipecac | gr. iv | \|26 |
| | Pulv. rhei | gr. xv | 1\| |
| | Sod. bicarb. | ʒiss | 6 |
| | Aq. menth. piperit. | q. s. ℥iv | 125 —M. |

Sig.—Shake. One to two teaspoonfuls (4.0–8.0) three times a day in water after meals as a mild laxative.

| | | |
|---|---|---|
| ℞. | Pulv. rhei<br>Magnes. usta<br>Sod. bicarb. | āā ʒiv (16.0).—M. |

Sig.—One-half teaspoonful (2.0) three times a day after meals.

| | | | |
|---|---|---|---|
| ℞. | Pulv. aloes | gr. xx | 1\|3 |
| | Ext. belladonna<br>Ext. nucis vomicæ | āā gr. v | \|3.—M. |
| Ft. pil. No. xx. | | | |

Sig. One to two pills at night.

Podophyllin combinations are quite useful.

| | | | |
|---|---|---|---|
| ℞. | Podophyllin<br>Ext. physostigmatis<br>Ext. nucis vomicæ | āā gr. v | \|3.—M. |
| Ft. pil. No. xxx. | | | |

Sig.—One pill at night and in the morning if required.

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[1] Purgen tablets (Bayer), each tablet contains 1½ grains (0.1) of phenolphthalein; each phenolax wafer contains 1 gr. of the same.

[2] Münchener med. Wochenschr., No. 41, 1903.

| | | |
|---|---|---|
| ℞. | Pil. colocynthi comp. | āā gr. j (0.065). |
| | Pil. rhei comp. | |
| | Ext. hyoscyam. | gr. ss (0.032).—M. |

One pill.

Sig. One pill before dinner.

| | | |
|---|---|---|
| ℞. | Aloin | gr. $\frac{1}{5}$ (0.013) |
| | Strychnin sulph. | gr. $\frac{1}{60}$ (0.00108) |
| | Ext. belladonna | gr. $\frac{1}{8}$ (0.008) |
| | Pulv. ipecac | gr. $\frac{1}{16}$ (0.004).—M. |

One pill.

Sig.—One to two pills at bedtime.

| | | |
|---|---|---|
| ℞. | Resinæ podophyllin | gr. $\frac{1}{6}$ (0.011) |
| | Pil. rhei comp. | gr. iiss (0.162) |
| | Ext. hyoscyam. | gr. $\frac{1}{2}$ (0.032).—M. |

One pill.

Sig.—One to two pills at night.

| | | |
|---|---|---|
| ℞. | Ext. colocynth comp. | gr. j (0.065) |
| | Ext. jalap | gr. $\frac{1}{2}$ (0.032) |
| | Resin podophyllin | gr. $\frac{1}{4}$ (0.016) |
| | Leptandra | gr. $\frac{1}{2}$ (0.32) |
| | Ext. hyoscyami | āā gr. $\frac{1}{4}$ (0.016) |
| | Ext. taraxaci | |
| | Ol. menth. pepmt. | q. s.—M. |

One pill.

Sig.—One to two pills at bedtime.

| | | |
|---|---|---|
| ℞. | Aloes | āā gr. $\frac{1}{40}$ |
| | Resin of jalap | |
| | Resin of scammony | |
| | Turpeth root | |
| | Extract of belladonna | āā gr. $\frac{1}{4}$ |
| | Extract of hyoscyamus | |
| | Almond soap | q. s.—M. |

Ft. pil. No. i.

Sig.—One to two pills on retiring.

An excellent combination of saline laxatives is—

| | | |
|---|---|---|
| ℞. | Sodium sulphate | ʒiij |
| | Magnesium sulphate | ʒj.—M. |

Ft. pulv. No. i.

Sig.—Dissolve in a half glass of lukewarm water, add a quarter of a glass of seltzer water, and drink at once.

Compound jalap powder, 30 grains (2.0), with calomel, 5 grains (0.6), is a good combination.

Jalap and colocynth belong to the stronger remedies, and I only employ them temporarily to empty the bowels. The same is true of castor oil and calomel.

Hunyádi, Friederickshall, the Homburg Waters, Carlsbad salts, Pluto, Apenta, Rubinat, Congress, etc., may be necessary for a brief period, but should not be used for any length of time. In anemic patients with constipation the following pills are of service:

| | | |
|---|---|---|
| ℞. | Pill (Blaud's iron) | gr. v (0.324) |
| | Aloin | gr. $\frac{1}{20}$ (0.032).—M. |

One pill.

Sig.—One to two pills three times a day after meals.

or

| | | |
|---|---|---|
| ℞. | Blaud's iron pill | gr. x (0.6) |
| | Pulv. capsici | gr. $\frac{1}{4}$ (0.016) |
| | Aloini.<br>Strychnin sulph.<br>Acid. arseniosi | āā gr. $\frac{1}{30}$ (0.0022).—M. |

One pill.

Sig.—One pill three times a day after meals.

*Fecal Colic, Fecal Tumor.*—It is an error to at once administer large doses of cathartics, and in some cases positive harm may result. The rectum should first be examined, and all material found therein removed by the finger and then by enemata. High injections of soapsuds and water, in all, 1500 c.c., containing olive oil 8 ounces to 1 pint (250–500 c.c.), should be given, in some cases in the knee-chest posture, in order to soften and remove accumulation. Frequent injections and irrigations should be given to start movement for the first twenty-four to forty-eight hours. Ox-gall, 1 to 3 drams (4.0–12.0), with glycerin, 2 to 4 ounces (60.0–125.0), added to the enema, are of value.

Olive oil, 2 to 6 ounces (60.0–200 c.c.), can be given by mouth, if necessary, t. i. d. to soften the dejecta. Later, castor oil, laxol (a tasteless castor oil), calomel, or compound jalap by mouth, and saline cathartics.

Frequent irrigations, in some cases with electricity, can be added to the treatment. Large doses of tincture of belladonna and strychnin may later be of service. In some cases it takes several weeks for an old accumulation to be completely removed.

*Spasm of the Sphincter.*—This should always be examined for, especially in cases of spastic constipation. Gradual dilatation, or, preferably, rapid dilatation under an anesthetic, are curative. Local disease should be treated.

## DIARRHEA

Clinically, diarrhea may be defined as abnormal rapidity of intestinal peristalsis, accompanied by frequent evacuations of the bowel contents, which are too liquid or are watery in character.

Some patients normally pass solid dejecta several times a day, but this is not diarrhea; yet a single solid movement may possess pathologic significance.

Diarrheal stools are caused by the excess of water in the feces, and may be due to the liquid contents of the small intestine being so rapidly hurried into the colon that little absorption is able to occur in the small intestine. Free transudation of water from the blood-vessels or the glands may be a factor.

Rapid peristalsis in both the small and large intestines, or in the latter alone, is another cause.

At times increased peristalsis is the only factor, and there are no chemic or physical changes in the bowel contents and no structural changes in the wall of the gut. Increased peristalsis usually involves the large intestine as well as the small.

Pathologic increase of intestinal peristalsis may be produced in numerous ways. In the majority of cases it is caused by intestinal diseases in which anatomic changes are present, as in intestinal catarrh, ulcers, typhoid, etc. It may be present without any apparent anatomic lesions, as a result of irritants in the contents of the bowel; or when the contents are normal, but the irritability of the nerves of the intestinal wall is increased; or when the muscular coats of the intestines are stimulated by an irritant circulating in the blood or affecting the central nervous system.

Frequently there are several factors. The appearances of the evacuation, both macroscopic and microscopic, in diarrhea vary according to the etiology of the disease and the anatomic changes in the gut, when such are present.

In every diarrhea it is important to know whether it is produced by abnormal transudation or exudation, with increased peristalsis of the large intestine; or whether, in addition, the *peristalsis of the small intestine is increased.* In the last event large quantities of unchanged digestive fluids and undigested food remnants are evacuated, and nutrition is markedly impaired. Bile-pigment reaction in the feces shows involvement of the small intestine.

We may classify two forms of diarrhea: first, with intestinal lesions; second, diarrhea without lesions. The first group is described in the appropriate sections. The second group, with no intestinal lesions, is classified as follows:

1. Diarrhea due to irritation from the bowel contents. Diarrhea dyspeptica, Diarrhea gastrica, Diarrhea stercoralis, and Diarrhea entozoica are subdivisions.

2. Diarrhea due to irritants transmitted in the blood, such as the uremic type.

3. Diarrhea nervosa (nervous diarrhea), due to irritation of the nervous system.

4. **Diarrhea Cathartica.**—This type belongs in a class by itself. It is thus referred to by Nothnagel, and merely mentioned in passing. Colocynth and aloin in excess may also produce the condition. The majority of purgatives stimulate the peristaltic action of the entire intestinal tract. The peristaltic action of the large intestine is chiefly affected, as by the aromatic laxative drugs. The movements in this case are thin and liquid, since increased peristalsis interferes with the absorption of the ingesta and intestinal secretions.

With the alkaline laxative salts the action is not only to increase peristalsis, but they withdraw the water from the blood and stimulate the intestinal secretions. The prolonged use of drastic purgatives or excessively large doses produce an acute catarrh of the intestines.

### DIARRHEA DUE TO IRRITATION OF THE BOWEL CONTENTS

**Diarrhea Dyspeptica.**—Certain articles of diet may produce diarrheal evacuations, such as fresh fruit, cucumbers, pickles, cabbage, turnips, beets, etc. Patients vary as to susceptibility. Milk produces diarrhea in some, while others it constipates. Excess of food or too great ingestion of water or beer with the food may prevent gastric digestion. The ingesta entering the intestines unchanged may cause diarrhea.

Intestinal fermentation or putrefaction, spoiled food, and autointoxication may produce diarrhea. With the last the diarrhea is due to more than the mere local irritation.

In neglected or severe cases of pure dyspeptic diarrhea, long-continued irritation may give rise to true catarrh.

**Diarrhea Gastrica.**—Einhorn and Oppler first called attention to diarrhea resulting from disturbances of the stomach functions.

Hypochlorhydria and achylia gastrica are the most frequent causes of this type of diarrhea; *more rarely* hyperchlorhydria or motor insufficiency. With hypochlorhydria or achylia, diarrhea with intestinal symptoms, such as flatulence, borborygmi, and colicky pains, may predominate. The stools are often quite undigested.

These cases, if prolonged, may develop intestinal catarrh.

**Diarrhea Stercoralis.**—Diarrhea with constipation. If constipation occurs in a person whose bowels usually are regular, diarrhea may follow the attack of constipation. The diarrhea is accompanied by colicky pains, bloating, and by the development of more or less offensive gases, such as sulphuretted hydrogen, etc.

It is believed that the diarrhea is caused by the development of these gases in the intestinal contents as a result of stagnation of the fecal matter. Hardened fecal matter may irritate the mucosa and produce secretion and peristalsis. With stercoral diarrhea the passages are at first formed, then mushy, and finally liquid. Scybalæ may be found in the dejecta.

The passage of flatus affords temporary relief. Thorough evacuation of the bowels relieves all the symptoms. Neglected cases may cause intestinal catarrh.

**Diarrhea Entozoica.**—Intestinal parasites, the tapeworm, for example, may in some cases cause persistent diarrhea. Like other types of diarrhea, there are probably at first no changes in the mucosa, but long-continued irritation will produce catarrh.

### DIARRHEA DUE TO IRRITANTS TRANSMITTED IN THE BLOOD

Diarrhea due to the hypodermic injection of certain drugs belongs to this class. The diarrheas of septicemia, nephritis, diabetes, cholera, malaria, etc., are explainable by this theory.

## DIARRHEA NERVOSA (NERVOUS DIARRHEA)

This type depends on nervous disturbances, without any morbid changes in the walls of the intestines. Trousseau first described nervous diarrhea. No impairment of digestive functions is present.

It originates either from excessive stimulation of the nerves governing peristalsis (the motor function) or from the transudation of serous material into the intestinal canal (secretory function), produced by nervous influences. In some cases probably both factors are concerned.

The stimulus may arise from the nerve-centers and be transmitted through the fibers of the vagus, sympathetic, or splanchnic nerves to the intestinal ganglia.

As an example of nervous diarrhea, numerous watery evacuations may occur as the result of some emotion, such as fright or shock, in which cases the stimulus arises in the brain centers. These are more especially acute transitory attacks.

Nothnagel and Peyer[1] report instances of chronic nervous diarrhea: thus, some persons will be attacked with gurgling, abdominal pain, tenesmus, and diarrhea as soon as they find they can secure no access to a water-closet; while with others the sight of the toilet produces diarrhea. Some patients may have attacks at definite hours, without any relation to surrounding conditions.

In others, nervous symptoms precede the diarrhea, such as vertigo, stupor, giddiness, congestion of the head, reddening of the face, hot flushes over the body, fear, oppression, palpitation, rapid breathing, etc. These symptoms often disappear after a few diarrheal movements.

The number of stools varies; these may be from two to four, or even to fifteen, consisting of thin liquid contents, with mucus rarely present. At times the first movement is solid, the next mushy, and the subsequent movements liquid.

Occasionally peristaltic unrest, borborygmi, and severe tenesmus may accompany the movements.

This form of diarrhea is found as a symptom in hysteria or neurasthenia, in the nervous and debilitated, and even in healthy persons after a nervous shock.

With Graves' disease and migraine this type may occasionally occur.

Charcot[2] describes attacks with tabes (intestinal crisis). Peyer speaks of a reflex form of nervous diarrhea found in consequence of abnormal processes in the genito-urinary tract; for example, in uterine catarrh, emissions, spermatorrhea, and sexual excess.

Fischl cites a case of diarrhea which persisted for several years and resisted all treatment. Replacement of a reflexed uterus cured the case.

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[1] Wiener Klinik, 1893, Heft 1.

[2] Prager med. Wochenschr., 1891.

Vicarious diarrhea in pregnant women of the neuropathic type has been described by Condio. The diarrhea takes the place of vomiting.

Nervous diarrhea has also been attributed to excessive smoking.

### DIARRHEA FROM EXPOSURE TO COLD AND WET

This occurs after a sudden or severe chill from exposure to cold, or wetting of the surface of the body, especially the feet or abdomen. Probably it is due to reflex irritation, transmitted from the cutaneous nerves.

Accelerated peristalsis of the intestines occurs, whether due to reflex stimulation or secondary to hyperemia, it is uncertain. This type of diarrhea is usually transitory. At times it may assume the character of true intestinal catarrh.

### TREATMENT OF DIARRHEA

The method of treatment depends on the cause. In the cases with anatomic lesions in the intestines, regulation of the diet and medication appropriate to each special type should be carried out. These methods are described under their special sections, such as under Intestinal Catarrh, Dysentery, etc.

With diarrhea due to a laxative, heat to the abdomen and opium are indicated. Pilulæ opii, 1 grain (0.065), three or four times a day, or one of the other opium preparations, or,

| | | | |
|---|---|---|---|
| ℞. | Tinct. opii | ʒiiss | 10 |
| | Bismuth. subnit | ʒiij | 12 |
| | Mist. cretæ | q. s ℥iv | 125.—M. |

Sig.—Shake. Two teaspoonfuls (8.0) four times a day.

With **dyspeptic** and **stercoral diarrheas** thorough removal of the sources of irritation are indicated, such as the use of calomel or blue mass, 5 grains (0.3), castor oil or laxol, 1½ ounces (45.0), or a saline cathartic, such as magnesium sulphate, ½ to 1 ounce (15–30 grams), or Sprudel salts, apenta, etc. Intestinal irrigation is indicated in these types.

For **intestinal parasites** an appropriate remedy and a cathartic should be administered.

**For Diarrhea Gastrica.**—Treatment should be given for the existing condition in the stomach; for hypochlorhydria, stomachics, dilute hydrochloric acid, intragastric faradization, etc.

For **achylia gastrica**, chiefly vegetable food, finely divided, and the methods employed for this condition.

For hyperchlorhydria, a rare cause, diet and the alkalis, etc., are indicated.

**For Diarrhea Due to Irritants Transmitted in the Blood.**—

The cause should receive treatment, thus, nephritis, malaria, etc. The general condition should be improved.

Heat locally to the abdomen, liquid diet, rest in bed, the bismuth and astringent preparations, such as bismuth subnitrate, 30 grains (3.0) t. i. d., bismuth salicylate, 10 grains (0.6) t. i. d., or bismuth subgallate, 10 grains (0.6) four times a day; or tannalbin, tannigen, or tannopin, or tanocol, 10 grains (0.6) each, three or four times a day.

Opium preparations may be required, but they should be used with caution.

**Nervous Diarrhea.**—If this depends on reflex action, such as from uterine disturbance, etc., the primary affection must be treated.

In other cases the general condition of the patient must be built up. Neurasthenic and hysteric conditions must receive special treatment. Constipating food may be administered.

Iron preparations, such as iron tropon, 1 to 2 drams (4.0–8.0), t. i. d.; peptomangan (Gude), 1 dram to $\frac{1}{2}$ ounce (4.0–16.0), in water t. i. d.; or Fowler's solution of arsenic, 5 minims (0.296 c.c.), or smaller doses t. i. d.; or sodium arsenate, $\frac{1}{50}$ to $\frac{1}{25}$ grain (0.0013–0.0026), are of value as tonics.

| | |
|---|---|
| ℞. Blaud's iron pill | gr. v (0.6) |
| Sodium arsenate | gr. $\frac{1}{50}$ (0.0013).—M. |

One pill. Make 30 such pills soft with honey and silver coat.

Sig.—One three times a day after meals.

The bromids of sodium, ammonium, or potassium, or bromid of strontium, given for a few weeks, 15 to 30 grains (1.0–2.0) t. i. d., lessen irritability.

Bismuth subnitrate or salicylate, in dosage already given, silver nitrate, $\frac{1}{8}$ to $\frac{1}{4}$ grain (0.008–0.016), and the astringents noted above are useful. Heat should be applied.

Opium and its derivatives are generally recommended for this, as well as other types of diarrhea, and are preferable to morphin.

The genral tendency to at once prescribe opiates in all diarrheas is to be deplored, especially in the nervous type, as the habit is readily gained. If other remedies fail, they may be used with caution. The following (Wm. H. Thomson) is a useful combination for such purposes. The dosage is small:

| | |
|---|---|
| ℞. Pulv. opii<br>Silver nitrate | āā gr. v (0.3) |
| Resin of turpentine | ʒij (8.0) |
| Liquor potass. | ʒj (4.0) |
| Pulv. licorice | q. s. to make pills soft.—M. |

Divide in pil. No. lx.

Sig.—Two or three pills three times a day.

| | |
|---|---|
| ℞. Tinct. opii camphor<br>Bismuth subnit. | āā ℥ss (16.0) |
| Mist. cretæ | q. s. ℥iv (125 c.c.).—M. |

Shake.

Sig.—Two teaspoonfuls in water every two or three hours.

℞. Tinct. opii.................................ʒiij (12.0)
Tinct. catechu comp.......................℥ss (16.0)
Aq. destil.........................q. s. ad. ℥iv (125 c.c.).—M.
Shake.
Sig.—Two tesapoonfuls in water every three hours.

Opium pills or other combinations can be employed.

For nervous diarrhea the intestines should be trained in the normal direction. Suggestion by the physician is of value. The patient should be instructed that after his morning evacuation he should refrain from other movements except when absolutely necessary. Often he can thus control the desire.

# CHAPTER XXIV

# INTESTINAL CATARRH; ENTERITIS; COLITIS; CATARRHAL SIGMOIDITIS; PROCTITIS; PHLEGMONOUS ENTERITIS

## ACUTE AND CHRONIC INTESTINAL CATARRH

(*Synonyms.*—Enteritis; Catarrhus Intestinalis.)

INTESTINAL catarrh is one of the commonest conditions with which we have to deal, and is of importance, since in acute cases in the young or aged it may seriously endanger the life of the patient; while the chronic cases are often obstinate and difficult to cure, and may impair the general health.

It occurs in two types, the acute and chronic, and these in turn may be primary or secondary to some other disease.

## ACUTE INTESTINAL CATARRH

(*Synonyms.*—Enteritis Acuta; Catarrhus Intestinalis Acutus; Cholera Nostras; Acute Diarrhea.)

Acute intestinal catarrh is defined as an acute inflammation of the intestines, characterized by pains of considerable severity and accompanied by diarrheal movements containing an admixture of mucus. The disease may attack a portion of the bowel and we may have a duodenitis, jejunitis, ileitis, colitis, sigmoiditis, and proctitis. In many cases the entire intestinal tract is involved. Appendicitis is described in a separate chapter. Though Woodward[1] held that the small intestine may not be involved alone, yet it unquestionably occurs. In many cases acute catarrh of the colon, on the other hand, gives the prominent symptoms, though there is frequently involvement of the ileum. The *inflammation may also be confined to the large intestine.*

**Etiology.**—*Age.*—It may occur at all ages, and is frequently found in infants and children. Acute intestinal catarrh may be primary (idiopathic) or secondary to some other disease.

*Primary acute catarrh* is due to the following causes: 1. An excessive quantity of food, so that a considerable portion remains undigested and acts as a source of irritation; heavy and indigestible food; extremely cold drinks, or an idiosyncrasy to certain foods. In others, who are suffering from slight intestinal disturbances, some articles of diet which otherwise would produce no difficulty may lead to the development of catarrh. Unripe fruit, tainted meat, milk or fish, or vegetables that are overripe or spoiled. Autointoxication with diarrhea may result from ingestion of such material, and catarrh is frequently produced if the source of irritation is not immediately removed.

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[1] Medical and Surgical History of the War of the Rebellion.

2. Chemic irritants, both organic and inorganic substances, such as colocynth, croton oil, jalap, senna, podophyllin, spices, pepper, copaiba, mustard, garlic, cantharides, mercury, arsenic, lead, copper, tartar emetic, phosphorus, antimony, alcohol, chloroform, ether, and some of the alkaloids, such as colchicin, veratrin, acids, and alkalis. With concentrated irritants the mucosa may be permanently damaged. Catarrh is caused when smaller quantities are ingested.

3. Mechanical irritants, such as hardened scybalæ, enteroliths, biliary calculi, foreign bodies, such as seeds, fruit-pits, coins, etc.

4. Exposure to cold or high temperature, especially in children and infants, or sudden variation in temperature, wetting the feet, are predisposing causes; these conditions probably favor the development of micro-organisms and the production of circulatory changes.

5. Chemic irritants from the blood, such as in catarrhal nephritis, catarrh from mercurial inunction, or from abdominal burns.[1]

Acute intestinal catarrh may be secondary:

1. To general infection, as in typhoid, dysentery, cholera, sepsis, influenza, pneumonia, scarlatina, measles, malaria, rheumatism, or other infectious diseases.

In dysentery and typhoid the ulcerations are in part responsible.

2. Direct action of micro-organisms, as in infantile catarrhs due to the activity of numerous types, such as proteus vulgaris and streptococci, Bacillus enteritidis sporogenes,[2] colon bacillus, also Bacillus dysenteriæ; and myiasis (larvæ of flies).

3. Extension of the inflammatory process from adjacent parts, as in peritonitis, invagination, hernia, tubercular or cancerous ulceration, and thrombosis.

4. Diseases of the liver, heart, and lungs due to stasis and engorgement.

5. In the cachexia of cancer, profound anemia, diabetes, Addison's and Bright's disease, intestinal catarrh may be a terminal event.

**Morbid Anatomy.**—The entire gastro-intestinal tract may be involved or only portions of the intestines are affected. These differences depend on the extent of the catarrhal process and upon the cause and intensity of the inflammation. The anatomic changes are not always commensurate with the severity of the symptoms.

The mucous membrane of the intestines is reddened uniformly or in spots, from light red to dark purple in color, especially marked around the follicles and plaques, on the summit of the valvulæ conniventes and of the villi. If the inflammatory process is intense, extravasations of blood occur. The mucous membrane is swollen and edematous, and is often covered with tenacious glassy mucus, stained by bile or blood and more or less opaque. Desquamated epithelial cells and occasionally a few pus-cells are seen in the mucus under the microscope; layers of epithelium may desquamate and form gray shreds. Fecal contents are usually liquid. The villi and solitary follicles are swollen and appear as whitish nodules surrounded by a red injected area (enteritis follicularis seu nodularis).

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[1] Elimination of the poisons through the bile and from the blood during intestinal secretion is the probable cause of the catarrh.

[2] This was believed to be possibly an impure culture of the Bacillus aërogenes capsulatus, though it may be a distinct organism (Herter, Bacterial Infections of the Digestive Tract).

If the process continues, these nodules may rupture and give rise to follicular ulcers. Catarrhal ulcers are produced by loss of the epithelial covering and extension of the inflammation. Irritation in the neighborhood of these ulcers may in protracted cases give rise to polypoid growths.

*Microscopically*, there are congestion and distention of the blood-vessels of the mucosa and submucosa and small extravasations are at times seen between the glands of Lieberkühn. The spaces between the glands are frequently widened and contain abundant masses of round cells. They are also present in the superficial or deeper layers of the submucosa.

The swelling of the solitary follicles is due to proliferation of their cells and to round-cell infiltration. This is also true of Peyer's patches when they are involved, which is rare to any extent. The epithelium of the mucosa is detached, especially in the large intestine, but this is believed to be chiefly a postmortem change.

The epithelium is undoubtedly involved in the catarrhal process, as degenerated epithelial cells are found in the mucus with the stool. The cells of the glands may be cloudy and swollen.

*Crypts of Lieberkühn.*—The glands are enlarged or the fundus is wider than normal, the opening is narrow, so that the crypt becomes bottle shaped. They may be detached from their base and raised, or protrude into the intestines, or even desquamate.

The submucosa is hyperemic. The muscular and serous coats are unaffected.

**Symptoms.**—They depend on the etiology, the location of the catarrh, and its severity, so that considerable variation occurs. The general symptoms of an ordinary primary attack are as follows:

It usually begins with a feeling of fulness in the lower part of the abdomen, with attacks of colicky pains and diarrhea. Nausea and vomiting may be associated with these symptoms at the incipiency of the attack. In the mild cases there may be no temperature, or it may be moderate, while in the severe types there may be a chill with rise of temperature to 103° to 104° F. In some cases there may be tympanites. There are gurgling sounds (borborygmi) and the abdomen is tender on pressure, at times over special regions markedly so. Loss of appetite occurs.

The number of stools depends upon the severity of the case. There may be only two or three movements in twenty-four hours, or as many as fifteen to twenty evacuations. The first one or two movements usually contain fecal matter and are somewhat mushy in character. They rapidly become semifluid, and finally thin and liquid. Feces and scybalæ may be found later. The more the colon is involved, the greater is the diarrhea. Diarrhea does not always occur if the small intestine alone is the seat of inflammation. The early stools are frequently of a dark brown color, sometimes of offensive odor, the latter condition being especially noticeable if dietetic errors are the cause.

The amount of material passed exceeds the quantity of food ingested, due to the digestive secretions and catarrhal secretion, all of which are rapidly evacuated. When the stools consist of watery discharge and mucus there is often little or no odor. They are slightly acid in reaction and foam-like in appearance.

The stools may be light yellow in color, grayish, or even greenish in young children, or colorless, resembling rice-water. These differences in color are dependent upon the location of the catarrh. In the yellow fecal material Gmelin's reaction for bile-pigment can often be obtained, and this is also found in the green movements, demonstrating involvement of the small intestine. The colorless stools most frequently occur in the specific choleraic types.

With duodenitis alone or associated with gastritis, when there is considerable duodenal catarrh and interference with the escape of bile, there may be jaundice, the presence of bile, and indican in the urine, at times albumin and casts, and the whitish stools found with jaundice.

Mucus is contained in the stools. It may float on top of the dejections in shreds of various sizes and be of glassy appearance, or be stained in various colors, or mixed with the bowel contents and form a jelly-like mass; it may coat the feces or be mixed with it in small amounts; or the movement may consist chiefly of mucus. In some cases the mucus may only be determined by the microscope.

The localization of the seat of the catarrh is shown by the characteristics of the mucus, to be described later.

*Microscopically*, there are epithelial cells, numerous micro-organisms, mucus, occasionally a little pus and blood, and undigested food particles in the stool. Blood is found only in severe cases where there is marked congestion or ulceration, and pus when ulceration is present.

*Chemically*, peptones and sugar may be present in the dejecta.

*Macroscopically*, food remnants may be seen with the naked eye for several days, especially if dietetic indiscretion be a factor.

*Subjective Symptoms.*—In the milder cases, except for the colicky pains, diarrhea, and the feeling of pressure and fulness, the patients may not feel very badly. In more severe cases they may feel chilly, feverish, dizzy, and weak, at times nauseated, and in some cases they may vomit. Tenesmus may be present if the lower part of the colon or rectum are affected. Gas may be expelled. Borborygmi may be audible. With children and elderly persons the symptoms are often pronounced. Collapse may occur. With infants the hydrencephaloid condition may occur, temperature 104° F. or more, sunken fontanels, rapid pulse, cold extremities, collapse, etc.

**General Physical Signs.**—The abdomen may be bloated, but when gas is expelled the distention lessens or disappears. Splashing sounds can often be elicited. Over the abdomen usually there is tenderness on palpation, especially in the region of the navel, and frequently in the right or left iliac regions, or along the course of the transverse colon; gurgling sounds can often be heard on palpation. In thin subjects peristaltic movements of the small intestine may be visible either before or after palpation.

If there is much gas, there is a tympanitic note on percussion; it may be dull in character if much fluid be present. Large accumulations of gas are not frequent.

**Fever.**—There may be no temperature or only moderate fever. In severe types the temperature may be quite high (102° to 104° F.). In some cases there are chills associated with the fever, but the temperature has a tendency *to fall after a few days* and does not show *the characteristic steady increase* of typhoid fever. With tainted food or bacterial infection, fever is especially apt to occur. Such cases run an acute course with severe clinical symptoms. Undoubtedly auto-intoxication is a prominent factor in their production.

With *gastroduodenitis*, *jaundice* is present, and often vomiting.

**Urine** may become scanty and concentrated, especially in severe cases where there are frequent movements, and there may be found cylindroids, albumin in small amounts, and hyaline casts. Indican is often present, especially if the small intestine is involved.

Rosenbach's reaction (Burgundy red), on boiling urine with nitric acid, is also found. This also shows intestinal putrefaction. Acetone has been found.

**Localization of Acute Catarrh.**—Involvement of the small intestine alone is more uncommon, and is usually associated with gastritis.

I believe in *acute cases* the involvement of the small intestine is a more frequent occurrence than some suppose; though the intensity of the inflammation may be greater *in one portion* of it, and in addition in some cases there may be an especially severe inflammation in the colon. Some believe the *large intestine alone is most frequently involved*. This is more so in *chronic cases*. In the rectum local involvement is quite frequent.

Acute catarrh of the caput coli, due to fecal accumulation, at times occurs, and this must be differentiated from appendicitis. The fecal tumor can be generally discovered by the doughy feel on palpation. The acute symptoms subside rapidly under intestinal irrigation and catharsis.

This refers to a simple catarrh and not to a true typhlitis which involves the muscular tissue. Acute catarrh may occur in the sigmoid and should be classified as *catarrhal sigmoiditis.* In sigmoiditis or perisigmoiditis the musculature is also involved. The cases described by Mayor and Leube are evidently of this type. Diverticulitis belongs to this last class. The nomenclature *should be very specific.*

*Localized Physical Signs.*—An *acute duodenitis* is usually associated with *acute gastritis,* and we have *jaundice with local tenderness* on pressure in the right portion of the epigastric region. Inflammation of the duodenum with local tenderness may occur after cutaneous burns.

Tenderness on pressure (pain), confined to the middle of the abdomen and not laterally, shows the affection to be probably confined to the other portions of the small intestine; but when the small intestine alone is involved, as diarrhea is usually absent, the diagnosis is difficult. The presence of a considerable number of undigested food particles and epithelial cells tinged with yellow bile-pigment in the feces; microscopic mucus mixed with the stool, with rarely a small amount of visible mucus, are a valuable aid to the diagnosis. Indican is usually present in the urine.

*Acute Colitis.*—With acute colitis[1] the pain and tenderness are most marked along the course of the colon, over the cecum, transverse or descending colon, sigmoid flexure, or over all together. The stools are diarrheal and contain considerable mucus.

Proctitis is characterized by *tenesmus* and colicky pains in the left iliac fossa. There is a constant desire to defecate. The scybalæ or stools are surrounded with mucus, sometimes tinged with blood, and the mucous membrane may prolapse during defecation and is red and tender. Rectal digital examination is accompanied by much pain, and the examining finger shows, at times, traces of blood.

The *most important method of diagnosis* to localize the process is by examination of the feces, noting the character of the mucus. Macroscopic examination is often sufficient.

When pure mucus is passed without any fecal admixture, catarrh of the rectum, sigmoid, or of the descending colon is indicated.

If small masses of fecal matter or solid balls are passed covered with a layer of mucus, the same condition is indicated.

If there is catarrh of the entire large intestine up to the cecum, even if the movements are thin, shreds of mucus are intimately mixed with the fecal matter, and can be recognized by the *naked eye.*

The close admixture of fecal material and mucus distinguish it from catarrh of the lower colon.

In *catarrh of the upper colon alone* or of the small intestine, or small intestine and upper colon alone, usually no mucus can be seen with the *naked eye*, and hyaline *microscopic* lumps of mucus are found intimately mixed with the stools. Small amounts mixed in the feces are at times visible.

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[1] More properly, acute catarrhal colitis, to distinguish it from dysenteric and other types.

*Diagnosis.*—If a colon-tube be introduced high into the rectum, and lavage be carried out intermittently with warm water through a funnel, by the same method as lavage of the stomach, the recovered fluid will contain visible mucus, and demonstrate that catarrh of the large intestine is present. This method was suggested by Boas.

The presence of yellow *mucous granules* in the movements have been considered diagnostic of inflammation in the small intestine, though Schmidt and Boas believe them to be structureless remains of muscle substance, casein, or egg-albumen, colored with bile-pigment.

*Bile-pigment.*—If the bile-pigment reaction can be obtained in the stool or in some of its constituents, this indicates an inflammation of the small intestine, and the more marked the reaction, the higher up the involvement. It may be found in the mucus, and this may be stained a dark orange, green, or greenish yellow.

Cylindric epithelium, round cells, or rarely fat (droplets) may be stained yellow.

An acid *reaction of the stool also shows involvement of the small intestine.*

Boas has subjected a filtrate of the feces to the digestion test with a small piece of albumin, and, when the result is positive, justly concludes that the condition originates in the small intestine.

**Duration.**—Mild cases may rapidly recover in three to five days, while severe cases often continue for several weeks. The intestines remain susceptible to irritation for a considerable time, and errors in diet may cause a recurrence of the attack. The condition may become chronic. Constipation may follow the acute attack, and this should carefully be treated, lest habitual constipation develop. The acute attack may never be entirely recovered from, but gradually develop into a chronic catarrh.

**Prognosis.**—These cases frequently recover within a short time, but in children or very old and enfeebled persons the disease may occasionally prove fatal. The prognosis as to cure depends upon the etiology of the disease; thus, if due to chemic irritants, the condition may become chronic.

**Treatment.**—*Prophylaxis.*—Particular articles of food or drink known to produce attacks of acute intestinal catarrh should always be avoided. Some are affected by ice-cream and ice-cold drinks, and these should be forbidden. Exposure to cold or wet should be avoided. *Rest in bed should be enjoined.*

When tainted food has been ingested or indigestible or an excessive amount of food, even though there is diarrhea, a laxative should be given immediately to thoroughly empty the bowel.

Calomel, 5 to 10 grains (0.3–0.6), followed by a saline cathartic, preferably within six hours for rapid effect, or castor oil or laxol, 1 to 2 ounces (30.0–60.0), should be given to an adult. Castor oil may be administered in coffee, sarsaparilla, ginger ale, or with orange or lemon juice.

For infants and young children calomel, $\frac{1}{2}$ to 1 grain (0.32–0.65), in divided doses, or 1 to $1\frac{1}{2}$ drams (4.0–6.0) of castor oil, or, preferably, laxol.

If an acid chemic irritant has been taken, then an alkali should be given, and vice versâ. Antidotes should be administered in the case of chemic poison. It is preferable also to administer a cathartic, so as to remove the poison from the intestinal canal.

Enteroclysis with normal saline solution at 110° to 115° F. is indicated in all these cases, employing 1 gallon (4 liters) by the recurrent method, so as to thoroughly cleanse the large intestine.

Calomel is useful when *there are flatulence and indicanuria.* I prefer a fairly large initial dose in these cases, and to repeat it within a few days, to the method of daily small doses. There is some danger of salivation from frequent small doses. Colonic irrigation once or twice a day is of great importance.

These methods, combined with salol or beta-naphthol-bismuth (orphol), 5 grains (0.3), three or four times a day, with the other bismuth preparations, or with urotropin, 5 grains (0.3), and sodium benzoate, 10 grains (0.6) t. i. d., are generally sufficient.

Dilute hydrochloric acid in 10-minim (0.59) doses t. i. d. is an excellent adjunct, providing there be no nausea or vomiting; oxyntin capsules with nux vomica may be substituted. Resorcin, 5 grains (0.3) t. i. d., may be employed for intestinal fermentation. One of the best remedies for diarrhea is bismuth.

Bismuth subnitrate, 20 to 30 to 40 grains (1.3–2.0–2.6), given four or five times a day, is of service. It may be combined with saccharated pepsin as a vehicle in mild cases in smaller doses; thus:

Bismuth subnitrate, saccharated pepsin, equal parts, $\frac{1}{3}$ to $\frac{1}{2}$ teaspoonful every two or three to four hours during the twenty-four hours.

Bismuth subcarbonate, 15 to 30 grains (1.0–2.0), four times a day.

Bismuth subgallate, 5 grains (0.3), three or four times daily with the bismuth subnitrate, is excellent.

Bismuth salicylate, 5 grains (0.3), four times a day is a good antifermentative; or ichthoform, 5 grains (0.3) t. i. d., in combination with bismuth subnitrate. Ichthalbin, 5 grains (0.3) t. i. d., is useful.

Tannalbin, tannigen, or tannopin, 5 to 10 grains (0.3–0.6), can be used in combination with bismuth.

℞. Bismuth. subnit.......................... ℥ss (16.0)
Mist. cretæ..........................q. s. ℥iv (125.0).—M.

Sig.—Shake. One to two teaspoonfuls in water every two or three hours. The same prescription, with 10 to 15 drops (0.6–1.0) of tincture opii camphor in each dram dose, is useful if the diarrhea continues excessive.

The following represent *single doses* of remedies which can be taken every three or four hours in persistent diarrhea:

| | | |
|---|---|---|
| ℞. | Tinct. opii | ♏x (0.59) |
| | Tinct. kino<br>Comp. tinct. catechu | āā ♏xx (1.18) |
| | Aqua destil | q. s. ʒij (8.0).—M. |

| | | |
|---|---|---|
| ℞. | Tinct. opii | ♏x (0.59) |
| | Mist. cretæ | ʒj (4.0) |
| | Comp. tinct. catechu | q. s. ʒij (8.0).—M. |

| | | |
|---|---|---|
| ℞. | Bismuth. subnit | gr. x (0.6) |
| | Tinct. opii deodor | ♏x (0.59) |
| | Aq. cinnamomi | q. s. ʒj (4.0).—M. |

I prefer, however, *to avoid opiates as much as possible in my treatment.*

Cotoin, 1 to 2 grains (0.06–0.1); tincture coto, 15 minims (0.888), or paracotoin, in double dose as compared to the cotoin, have been suggested for diarrhea.

Codein, $\frac{1}{8}$ to $\frac{1}{4}$ grain (0.008–0.016), or, rarely, morphin may be required. Patient should be kept in bed.

**Heat.**—*Dry* or *moist heat*, or Priessnitz compress, hot-water bag, flaxseed, or milk or potato poultice, hot pieplate, hot salt-bag, compress of hot water, pepper poultice, weak mustard and flour poultice should be applied. If there are symptoms of collapse, warmth, hot drinks, camphor hypodermics, and strychnin are indicated. Hypodermoclysis may be required.

Enteroclysis is useful to rid the bowel of irritating material, and for treatment of the acute catarrh when located in the colon. It may be given by enema or, preferably, with the recurrent tube. About 1 to 1½ quarts (1000–1500 c.c.) should be used by enema, hips elevated, injecting with a long tube.

When there is marked fermentation—

| | |
|---|---|
| Acetozone | 1 : 2000 to 1 : 1000; |
| Alphazone | 1 : 2000 to 1 : 1000; |
| Peroxid of hydrogen | ℥j (32.0) to 2 quarts (liters), or |
| Salicylic acid (1 : 1000) | 2 quarts (liters); |
| Boric acid | ʒj (4.0) to 2 quarts (liters); |
| Permanganate of potash | 1 : 3000. |

Irrigate with 2 or 3 quarts (liters) with a recurrent tube once a day.

Soothing irrigations are flaxseed tea, gum-arabic solution in water at 110° F., slippery-elm water, or normal saline solution.

Occasionally astringent irrigations may be necessary.

First wash the bowel with warm water, then inject tannic acid, 30 grains (2.0) to 1 liter (quart), and 15 drops (0.888) of laudanum, and hold a while. Weak nitrate of silver, 5 grains (0.3) to 1 quart (liter), has also been recommended, but is better in chronic cases. Gastritis, jaundice, and malaria should be treated, if present, or any disease to which the intestinal catarrh is secondary.

In referring to the *cases of duodenitis with jaundice*, the author wishes to impress on his readers the value of urotropin and sodium benzoate. This should be given in doses, 10 grains each, dissolved in water, by rectum, if acute gastritis is present, and in 5 grains (0.3) each, when this has subsided. These remedies have an excellent effect on the severe indicanuria which I usually found present, and the urotropin disinfects the biliary tract, and I believe prevents cholecystitis and other biliary infections. The writer always uses these methods. High enemata or irrigation with hot normal salt solution are of value. Occasionally cold enemata may be used. The sour milks are, moreover, of great value.

If acute gastritis accompanies the acute duodenitis, the former condition must receive particular attention, since remedies directed to the obstructive jaundice (duodenal) would not be retained. External heat should be kept on continuously, occasionally a mustard poultice, and soda bicarbonate given at first in small doses, 5 grains (0.3), to liquefy the bile. *When the acute gastric condition has subsided*, the soda bicarbonate can be increased to 15 grains (1.0), or more t. i. d., and later, salicylate of soda, 5 grains (0.3) in capsules, can be substituted t. i. d. for the soda. The following prescriptions aid intestinal digestion, and the sodium succinate has an excellent effect on the biliary tract. They represent a single dose in capsule:

| | | |
|---|---|---|
| ℞. | Holadin | gr. iij (0.198); |
| | Succinate of soda | gr. v (0.3); |
| | Bile salts (Fairchild) | gr. ss (0.033).—M. |

| | | |
|---|---|---|
| ℞. | Holadin | gr. ij (0.132); |
| | Bile salts | gr. ss (0.033); |
| | Phenolphthalein | gr. j (0.066).—M. |

| | | |
|---|---|---|
| ℞. | Bile salts | gr. j (0.066); |
| | Succinate of soda | gr. v (0.3); |
| | Phenolphthalein | gr. ss (0.033).—M. |

Sig.—One capsule before meals or at bedtime.

Olive oil, $\frac{1}{2}$ to 1 ounce (30.0–60.0) t. i. d., is of value when it can be retained. Oleic acid, 5 minims (0.3) in capsule, can be substituted.

*Diet.*—Cold drinks should be avoided. Sanatogen is useful.

Warm teas, such as chamomile, fennel, anise seed, or plain tea and gruels (barley or rice), with or without milk, very dilute milk with lime-water, etc., should be given. Milk is often indigestible and barley broths preferable.

Later, bouillon, water soup (bread softened in hot water with butter and salt), yelk of egg or white, or entire egg (raw or soft boiled), stale bread, boiled water, etc. Avoid *carbonated waters*. Vichy and milk may be given if the gas is allowed to pass off before drinking.

As soon as the diarrhea is over, soft-boiled eggs, calves' brains, scraped beef, mashed potatoes, cocoa, weak coffee, chicken, chops, steak, stale bread and butter, potatoes mashed or baked can be given. Fruit, green vegetables, hot breads, fat, and acids should be avoided for a considerable period.

## CHRONIC CATARRH OF THE INTESTINES; CHRONIC COLITIS

(*Synonyms.*—Enteritis Chronica; Chronic Enterocolitis.)

This disease is characterized by a chronic inflammation of the mucous membrane of the intestines, which gives rise to various functional disturbances of the bowels. Any portion may be affected.

**Etiology.**—Chronic intestinal catarrh may arise from an acute enteritis which shows no tendency to recovery, or more frequently from repeated attacks of acute catarrh which follow each other at short intervals before complete recovery occurs. Patients frequently pay no attention to an apparently mild attack and disregard the rules of diet prescribed. As a result, the condition becomes chronic.

In other cases, however, chronic catarrh may have an insidious onset from the beginning. Fecal accumulation, notably scybalæ, may be a cause of a subacute or chronic catarrh. Pressure of tumors narrowing the canal, as pressure from fibroids of the uterus or adhesions, may be factors.

The direct causes of chronic enteritis are the same as in the acute type, and chronic catarrh may be either primary (idiopathic) or secondary to other affections. For the complete etiology the reader should refer to the causes of acute intestinal catarrh.

Chronic catarrh may be secondary to diseases of the lungs, especially tuberculosis, and also diseases of the liver, heart, kidneys, and diabetes. Intestinal parasites, tapeworms, round-worms, seatworms, etc., may be causes by producing irritation. I recently attended a case of splanchnoptosis, in which there was marked prolapse of the sigmoid flexure with a tendency to fecal accumulation therein. The patient never had an acute enteritis, but for five years has had discomfort in this region, and invariably every few days the passage of strings of mucus and scybalæ. There were no symptoms of mucous colic. The case was one of chronic catarrh of the sigmoid. The symptoms disappeared under treatment for visceroptosis. The possibility of chronic localized catarrh from visceroptosis, associated with fecal accumulation, is worthy of consideration. Chronic appendicitis is suggested as a cause by G. R. Lockwood, and the cure of chronic colitis has followed appendectomy.

*The rectum should always be examined.* Pressure on the rectum from a uterine fibroid I have seen produce partial stenosis with coprostasis above and resulting intestinal catarrh. Ulcer, fissure, or hemorrhoids may cause not only local manifestations, but catarrh higher up. I have seen a case with chief symptoms pointing to the descending colon and sigmoid, in which an ulcer high in the rectum was the cause.

**Morbid Anatomy.**—The anatomic changes in chronic enteritis are similar to those in acute cases, being characterized by hyperemia, swelling of the mucous membrane, and increased secretion of mucus. The color of the mucosa varies from a dark venous red to a pale grayish-red tint; in some cases it may even be gray or slate colored from extravasation of pigment between the glands and at the tips of the villi. The last cases are those of long duration.

The surface of the mucous membrane is covered with a transparent viscid mucus and the epithelial cells are cloudy, in a condition of fatty degeneration, and partly desquamated. In the majority of cases of chronic catarrh the accumulations of round cells (which are characteristic of the acute type) are not seen, but *there is connective-tissue proliferation* in the chronic form.

*Exceptions.*—In some of the early cases of chronic enteritis the microscopic picture may be much the same as in the acute process; and in other mild cases the only abnormality determined is the accumulation of pigment between the glands or in the muscularis mucosæ and a slight widening of the interstices.

As a result of chronic enteritis, *hypertrophy or atrophy of the intestinal mucosa may develop.*

*Hypertrophy of the Intestinal Mucosa.*—The glands of the mucosa are elongated, tortuous, irregular in shape, and may form diverticula. Their orifices may become occluded through connective-tissue proliferation and there will be a retention of secretion and the formation of cysts (enteritis chronica cystica). Connective-tissue proliferation leads to the formation of polypi (enteritis polyposa), which are more rare, and are usually found in the large intestine. In many cases the walls of the intestines may become *thickened throughout,* including the *muscular coat,* to the extent of several *times its normal thickness.*

Woodward reports a few cases of proliferation of the intestinal mucosa with its glands.

*Atrophy of the Intestinal Mucosa.*—The clinical entity of atrophy of the intestines has been in considerable dispute, and undoubtedly a pseudo-atrophy due to postmortem change occurs.

Riegel, Ewald, and Einhorn believe there is a true intestinal atrophy. In view of the existence of an atrophy of the gastric mucosa resulting from a chronic gastritis and from its occurrence in pernicious anemia, I am convinced that an *atrophy of the intestinal mucosa* may occur in advanced cases of chronic intestinal catarrh. The condition I believe to be rare. Such atrophy may originate in the glandular tissue in the glands of Lieberkühn. There may be an infiltration of round cells, a fatty degeneration, a disintegration, and desquamation or atrophy. On the other hand, it may result from a connective-tissue proliferation compressing the glands.

Musgrave believes an atrophic condition of the intestines may follow the chronic catarrh occurring with amebic dysentery.

The villi degenerate with the atrophy of the glands, shrink, and become small. No ulceration of the solitary or agminate follicles occurs, and it is a question whether atrophy to any extent ever takes place in them.

There is a *degeneration of the muscular coat* and *some thinning* of it.

Jürgens[1] has described a fatty degeneration of Meissner's and Auerbach's plexus and of the muscular tissue, as a special type of intestinal atrophy; Sasaki records 2 similar cases dying with the clinical symptoms of pernicious anemia. These conditions probably are related to intestinal atrophy.

Atrophy of the mucosa occurs more frequently in the colon, especially in the cecum, ascending colon, or ileum near the valve. Large portions of the intestines are rarely found atrophied, but the process generally involves certain parts.

*Ulcerative Processes.*—As in acute enteritis, we may have *ulcerative processes in chronic catarrh of the intestines.* Superficial erosions of the mucosa may become deeper with the production of ulcers. Rarely they increase sufficiently in depth, and result in erosion of a blood-vessel with hemorrhage, or cause a local peritonitis with or without abscess, or even a perforative peritonitis. Generally the ulcers remain unchanged for a considerable time or cicatrize; occasionally stricture of the intestines may result. The follicles may occasionally swell up and burst, producing small follicular ulcers. Frequently healing takes place. *Extensive ulcerations are seldom met with in chronic enteritis, unless accompanying a tubercular process.*

The "sago grains" or frog spawn in the feces, formerly believed pathognomonic of follicular ulceration, are shown to be of vegetable origin.

Kitagawa finds that some of these grains are mucous in character, but this in itself militates against ulceration, as ulcers do not discharge mucus, but pus The presence of mucus merely indicates catarrh.

**Symptoms.**—The chief diagnostic symptom of chronic intestinal catarrh is the *abnormal character of the feces.* It seems advisable to first describe the subjective and objective symptoms which occur in many cases. Some patients complain of no subjective symptoms whatever. The majority of cases complain of a feeling of discomfort or occasionally of slight pains in the abdomen. These symptoms are apt to be most marked after eating, usually several hours; or frequently just before or even after defecation. At times these sensations may disturb the patient an hour or two before rising.

There is a feeling of tension or bloating of the abdomen which may be relieved by the passage of flatus, and this tendency gives rise to considerable annoyance. Flatulence may become so severe as to cause shortness of breath, an asthmatic attack, palpitation, angina pectoris, or cerebral congestion and vertigo, all of which symptoms are alleviated by belching of gas. Flatulence is much more characteristic of chronic enteritis than of the acute type.

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[1] Berlin. klin. Wochneschr., 1892, p. 357.

Borborygmi are often present. Severe pains are usually absent, though slight colicky pains of rather brief character may be present. These are often relieved by bowel movement or by the expulsion of flatus.

In some cases the general health is not impaired, while in other cases it is undermined. General nutrition may become impaired, especially if the small intestine is also involved. In the latter case gastric symptoms, such as anorexia and nausea, and occasionally vomiting, may be met with.

The patient may feel weak, disinclined to work, be irritable, depressed, and even hypochondriac or melancholic. There may be loss of weight, anemia, slow pulse, cold extremities, and attacks of severe headache. The nervous symptoms are due in part to depression from an evidently chronic and persistent disease, and in a large degree to auto-intoxication.

**Physical Signs.**—The abdomen may appear distended, especially a couple of hours after eating, with some tenderness on pressure. In chronic enteritis the colon seems to be more usually affected.

*Chronic Catarrhal Colitis.*—There may be *chronic catarrhal colitis*, with tenderness over the caput coli and ascending colon, with the sensation of a hard mass which yields to the examining finger on pressure (fecal accumulation), or this part may be tympanitic and give the splashing sound from gas and liquid. The same may be true over the descending colon, sigmoid flexure, or transverse colon. There is often tenderness on pressure along the entire colon. Pain is felt directly under the point of pressure, or occasionally at a different point further along the colon, due to the passage of gas, which has been forced along by local pressure.

In thin persons peristaltic movements of the intestines are occasionally observed, especially after palpation. In some cases there are no special objective symptoms.

The diagnostic symptom in chronic intestinal catarrh is the abnormal character of the stool—the *presence of mucus*.

The movements are irregular and the consistence of the stool is variable, *but the mucus is diagnostic*. Diarrhea is not a constant symptom.

There are four types of movements which occur in chronic enteritis:

1. In one set of cases there is marked constipation, and a solid movement occurs only once every two or three days and at times only after a cathartic. The feces are usually hard. It is probably due to a diminished activity of the automatic nervous mechanism of the intestines produced by the catarrh (Nothnagel).

2. In others, constipation and diarrhea alternate; there may be hard movements for several days and these succeeded by five or six

thin or mushy movements, mixed with mucus, and accompanied by severe colicky pains. These, in turn, will be followed by constipation, and so on. In some instances the evacuations will be fairly normal for several days and then diarrheal movements will occur, and after this constipation. Constipation is the chief feature in these cases. The reflex irritability of the nervous apparatus, however, is quite good, and decomposition of the stagnating bowel contents eventually causes increased peristalsis with diarrhea.

The periods of constipation or diarrhea, on the other hand, may continue for a long time; thus, constipation for several weeks or months, and then diarrhea for weeks or months. Probably in the latter class there is an acute exacerbation of the catarrh.

3. Rare cases occur in which there is a daily evacuation of unformed and mushy feces.

4. Cases in which for months the patients pass several diarrheal stools each day. The small intestine is involved as well as the large bowel, as there is a bile-pigment reaction, as a rule, or there are yellow fragments of mucus or epithelial and round cells tinged with bile. The food on account of the catarrhal process is not completely digested in the small intestine, and abnormal products, such as acids, etc., are produced, so that the undigested food and fermenting material give rise to increased peristalsis.

In addition, there are some in which the nervous element is a factor combined with the catarrh, and movements occur during the night or early in the morning. F. Delafield[1] describes this type.

The *presence of mucus in the feces is characteristic*. The type of mucus in mucous colic (Enteritis membranacea), which occurs in large amount, and the symptoms render the differential diagnosis comparatively easy. In other cases the presence of mucus demonstrates a true catarrh. In habitual constipation there may be a thin shellac-like covering of mucus over the scybalæ, and this appearance is not found in chronic enteritis. With chronic catarrh with constipation, the quality of the dejecta may be nearly normal, except there is an admixture of mucus. In rare cases the mucus may be absent, or it may be very tough and adhere to the intestinal wall, or the scybalæ may be too small to scrape it off. However, on most occasions, mucus will be present, and if there is doubt, washing of the bowel by means of the tube and funnel will eventually bring it away.

Besides the presence of mucus in mucous colic, in some cases of intestinal dyspepsia there is mucus in the stools.

| *Intestinal Dyspepsia.* | *Chronic Enteritis.* |
|---|---|
| Pure mucus *alone.* Stools gelatinous. Mucus microscopic and seldom visible. | Mucus with epithelial and round cells (diagnostic). |
| *Green stools with acid reaction;* bile-pigment gives pronounced reaction. | |
| No fecal odor. | Alkaline stools. Fecal odor. |

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[1] Med. Rec., May 11, 1905.

The quantity of mucus varies greatly; in most cases there may be only a small or moderate amount.

The various *combinations of mucus* with the stool and the *localization of the catarrhal process* have been described under Acute Enteritis, to which I refer my readers.

We may have therefore: (1) Chronic catarrhal enteritis (alone), rare; (2) chronic catarrhal enterocolitis; (3) chronic catarrh of various portions or of the entire colon or of the rectum; thus, chronic catarrh of the caput coli, of the ascending, transverse, or descending colon; or chronic catarrhal sigmoiditis, or proctitis. *The colon is most frequently involved.*

When the movements are watery and thin, the fecal matter is a light brownish yellow or grayish yellow, and may contain little biliary matter. Undigested meat or starch particles can often be seen in these cases.

*Microscopically.*—Though nothing may be discovered macroscopically, we may find with the microscope undigested meat-fibers, starch granules and fat droplets, also mucus and *round and epithelial cells*, at times yellow and shrivelled up. They indicate catarrh of the small intestine. Blood is never present unless due to ulcer or hemorrhoids. Pus is rarely found and only as isolated cells.

Dejecta resembling pus diluted with water (Blenorrhœa intestinalis) shows diphtheritic inflammation when pus is seen in large amount under the microscope. Large masses of epithelial cells in various degrees of degeneration are present in chronic catarrh. They are responsible for the cloudiness in the mucous secretion.

The character of the food and of the stool, and whether there is constipation or diarrhea, determine the consistence and reaction of the feces and the degree of fermentation. As a rule, alkaline reaction is present. The presence of fermentation and putrefaction can be determined by the abdominal tension, flatus, and character of the stool, which may be fetid and present a foamy surface.

Fecal material may be placed in a fermentation tube and kept at blood temperature for several hours, and the degree of fermentation or putrefaction will be shown by the quantity of gas in the tube. The method is described under Testing the Intestinal Functions.

The presence of putrefaction will be shown by indican in the urine, and the Rosenbach reaction (Burgundy red) on the addition of nitric acid and boiling.

A chronic catarrhal enteritis complicated with ulcers will show marked diarrhea with pus and blood in the stool. If the ulcers occur in the small intestine *alone* and there is no diarrhea, pus and blood often *will not be present*, but there will be more severe pain, more marked tenderness on pressure, and the clinical symptoms will be more severe. Occult blood should be tested for.

The diagnosis of the atrophic type of chronic enteritis is extremely difficult. Some question its possibility. There will be a previous history of intestinal catarrh of long standing. Later there will be diarrhea, no mucus, with gradual loss of weight, and at times symptoms of pernicious anemia. Tuberculosis must be excluded. This condition is more frequent in the young.

**Course.**—The course of chronic intestinal catarrh is usually very tedious. It may last for many years. There may be periods of perfect health, but there is a tendency to relapses from any slight indiscretion.

**Differential Diagnosis.**—The method of localization of the catarrh, as previously stated, is the same as in acute enteritis.

Irregular bowel action, with the presence of mucus in the stools of the *character described, associated with abdominal symptoms of discomfort, suggest chronic catarrh.* The discharge of mucous colic is characteristic, as are also the symptoms. In the mucus which is occasionally found with intestinal dyspepsia there is an absence of *epithelial and round cells,* and the stools are green and acid, as I have already stated.

In habitual constipation there is an *absence of mucus* in the movements. With marked fecal impaction I have frequently seen a small amount of mucus in the feces due to temporary irritation. After removal of the impaction and subsequent care of the bowels there is no further appearance of mucus. Impaction, if neglected or occurring in frequent attacks, may be a cause of local intestinal catarrh, and, as heretofore noted, occasionally of stercoral ulcers.

With malignant disease of the intestines, enteritis is often associated, but the cachexia is marked and other symptoms of the neoplasm are present. With intestinal ulcers there are marked pains, local tenderness, and pus and blood in the stool. With enteroptosis we may have a prolapse of the sigmoid, fecal accumulation, and chronic catarrhal sigmoiditis. In every case of chronic intestinal catarrh, *enteroptosis should be examined for.*

*Rectal examination should be made in every case of chronic intestinal catarrh,* as the focus occasionally starts from the rectum and progresses up the bowel as, for example, from an ulcer. On account of its importance I shall devote a brief section to Proctitis.

A uterine fibroid may block the bowel, acting as a ball-valve, and, from fecal accumulation above this point, a marked catarrh of the mucous membrane may result.

Chronic appendicitis may result from a chronic intestinal catarrh, while in some cases a chronic appendicitis may act as a focus of irritation for a localized chronic catarrh in the cecum. Removal of the appendix is curative in the last type. In certain diseases of the stomach there may be constipation or diarrhea, but the absence of mucus in the stools and examination of the gastric contents will settle the question.

In chronic enteritis of the small intestine alone there are usually gastric symptoms, constipation, yellow tinged mucus in the stools, generally microscopic and well mixed with the feces, and the biliary salts reaction.

With chronic catarrhal colitis there is *usually more constipation* and more or less mucus of a grayish tinge, either covering or on the surface of the feces or in small strings. Pure mucus may at times be voided at the end of defecation if the lower bowel is affected. Palpation is of value in locating the position of the chronic colitis, as the affected area is apt to be sensitive to pressure.

*Chronic diarrhea is prominent* in cases in which both the small and large intestines are involved, and the mucus may be yellow in color and considerable undigested food is often present.

**Prognosis.**—This depends on the severity of the symptoms, the duration of the disease, and the physical condition of the patient.

In the very young, very old, or those weakened by some other disease, such as endocarditis or tuberculosis, severe types of chronic catarrh may be accessory in producing a fatal result.

As a rule, the prognosis as to life is favorable, but in the severer cases of long standing it is not so favorable as to perfect cure.

The cases have a tendency to relapse, especially after indiscretions, though they may continue for a considerable time with comparative comfort. The milder cases of not long standing may recover under appropriate treatment.

**Treatment.**—The cause of the chronic catarrh should be carefully sought out and corrected. If, for example, endocarditis with resulting circulatory disturbances (which are a predisposing factor) be present, this should receive treatment. The Nauheim bath, even though taken at home by means of the Triton salts, would be of value in such cases. If enteroptosis is present, such as in the case I have described, where there was marked prolapse of the sigmoid, fecal accumulation, and local chronic catarrh, treatment by abdominal support (Rose's belt) and increasing the nutrition by the methods described under Gastroptosis would be indicated, in addition to the general treatment for chronic catarrh. I have seen one obstinate case cured by S. Gant by drawing up and suturing the sigmoid to the abdominal muscles. Angulations of the sigmoid should be corrected. If worms are present, they should be removed.

These remarks will sufficiently illustrate the necessity for investigation of the cause of the catarrh. Hygienic and dietetic measures are of great importance.

The patient should regulate his life carefully, not overwork nor be under too great business strain, eat slowly and at regular hours, and live in the open air as much as possible. In the constipated cases considerable exercise is of value. When the diarrhea is marked the patient should keep quiet during its active stage, and in some cases remain in bed until it has passed. Nervous disturbances should be avoided. The patient should exercise care not to wet the feet or undergo exposure to cold, and should be properly protected in rainy weather. A flannel band about the abdomen is of value. Change of scene and climate are often serviceable.

*Diet.*—The patient should eat at regular hours, slowly, and masticate thoroughly. It is preferable to give small meals more frequently than three large meals, and sufficient nourishment should be administered so that there is an increase in weight.

Fried foods, hot breads, rich pastries, desserts, and indigestible substances should be avoided. Though some allow the very moderate use of light wines, and occasionally beer or ale, in constipated cases, and the use of claret in diarrhea, in my own experience I find that patients with catarrhal conditions of the gastro-intestinal tract do much better by eliminating alcoholic beverages altogether.

The character of the diet is dependent upon whether diarrhea or constipation is present.

In *diarrheal cases*, if the type be severe, milk and lime-water equal parts, or boiled milk, or milk and barley-water, or rice-water in combination, often are efficacious, though some patients have an idiosyncrasy to milk. Rest in bed for a brief period may be necessary.

Koumiss, matzoon, bacillac, or lactone-buttermilk are often useful in such cases, though with some these preparations are objectionable, increasing the diarrhea. Sanatogen in 1-dram (4.0) doses, also raw eggs beaten in milk, may be of value.

In milder cases of diarrhea the diet may be more liberal. Carbonated waters, lemonade, fruits, salads, acids, cabbage, cauliflower, rye bread, brown bread, ice-cream, pastries, oatmeal, green vegetables, corn and beans, turnips, carrots, beets, radishes, celery, and lobster should be *avoided*. Mashed and baked potatoes, rice, sago, macaroni, bread well baked and toasted, with a moderate amount of butter, cream, soups, bouillon, soft-boiled or scrambled eggs, sweet-breads, calves' brains, chicken, lamb chops, lean fish, cocoa, tea and milk, or matzoon (with some) can be given.

The drinks should not be too hot or too cold, and an excess of liquid should be avoided.

In constipated cases the diet must be more liberal. In addition to the food mentioned above, fruits, such as oranges, ripe pears, grapes, green vegetables, such as spinach, peas, lettuce, cauliflower, plenty of butter, cream, and fluids are of service.

Cabbage, cucumbers, brown bread, sausages, lobster, mayonnaise dressing, and bran breads should be avoided in these cases also.

The *urine findings* should modify the diet—if indicanuria, less meat or none for a time; if fermentation, less vegetables; if nephritis, an appropriate diet.

*Massage.*—In cases *characterized by chronic constipation*, gentle massage over the course of the colon or the use of a light cannon-ball is indicated. No pressure should be exerted on the ball, but it should be rolled along the colon for five minutes morning and night. Light vibratory massage is of value under similar conditions.

*Hydrotherapy.*—Warm salt baths at 98° to 100° F., or Nauheim baths (artificial), eight to ten minutes every other day for two or three weeks, or pine-needle baths, bran baths, or mud baths may be of some service in some diarrheal cases. Cold baths should be avoided in diarrhea. A Priessnitz wet pack over the abdomen is of value when applied on retiring.

Cold showers and cold sponges are of service in nervous cases. Cold sitz-baths and cold douches over the abdomen are of use in constipated cases, but should be preferably carried out at some sanatorium. I have rarely found the latter methods necessary, but use the artificial Nauheim, pine-needle, or bran baths, also the cold compress to a considerable extent.

*Mineral Waters.*—A methodic course of drinking certain alkaline or saline mineral waters has proved beneficial in many cases. When taken at the springs, the patient is obliged to follow a rational method of life and diet and is free from worry, and thus receives additional benefit. Carlsbad is especially valuable when there is a tendency to diarrhea, and Vichy is next.

In cases of marked constipation, Marienbad is of service, and also the Hawthorne and Congress Springs at Saratoga. Virginia Hot Springs are also to be recommended. Where neither diarrhea nor constipation are prominent, Kissengen or Homberg; with constipation and anemia, Franzensbad and Elster.

Carlsbad water should be taken in small quantities, a wineglassful twice a day, or even in small amounts of the imported salts, 30 to 60 grains (2.0 to 4.0), three to five times a day. It is preferable to begin with small doses. Some do badly with this method. If the diarrhea increases so that the patient begins to lose weight, the treatment should be discontinued at once.

*Medication.*—The method of internal medication depends upon whether constipation or diarrhea is the existing condition. As a preliminary it is always wise to begin treatment with a thorough cleansing of the intestines by a single dose of castor oil, 1½ ounces (45.0), or a good dose of Carlsbad salts or magnesium sulphate.

If there be marked fecal accumulation, it is better first to employ enteroclysis or enemata, to remove the impaction, and then follow with the single cathartic.

In *constipated cases*, fruits, buttermilk, cold water (glass) on rising, stewed fruits, and a regular hour for attempted stool are all rational.

An enema, olive oil, 4 ounces (125.0), increasing to 1 pint (500 c.c.) or more, given slowly by a long tube on retiring, and to be retained, is an excellent procedure. It may be necessary to employ soapsuds enema (never over 1 quart) or normal salt solution. Rhubarb pills, fluidextract of cascara in 1-dram (4.0) doses, or the same quantity of aromatic fluidextract of cascara, regulin, or podophyllin pills are of sevice. Carlsbad water has been given by enema.

Small doses of castor oil or olive oil combined with salol are of considerable value in all cases. Thus:

Salol, 5-grain (0.3) tablet; with it give castor oil, 10 minims (0.59), in a gelatin capsule coated with shellac, four times a day; or olive oil can be substituted.

In the constipated cases larger doses of olive oil by mouth, 1 to 4 ounces (30.0–125.0) t. i. d., are of service.

The oil preparations seem to have an excellent effect on the mucous membrane. Lead and zinc preparations I do not employ.

Nitrate of silver is sometimes of service. It can be given in aqueous solution, each 1 dram containing $\frac{1}{6}$ to $\frac{1}{4}$ grain (0.011–0.016), being kept in a dark bottle, or the same dosage in an enteric coating.

The following pill, $\frac{1}{8}$ grain (0.008), of silver nitrate is excellent:

| | | |
|---|---|---|
| ℞. | Argenti nitratis | gr. v (0.3) |
| | Resin turpentine<br>Liq. potass. | āā ʒj (4.0) |
| | Pulv. licorice | q. s.—M. |

Div. in pil. No. xl.
Sig.—One pill t. i. d.

Bismuth salicylate, 5 to 10 grains (0.3–0.6) t. i. d., is of some value, but a movement should be secured every day *if it is given in the constipated cases.*

The bismuth and tannin preparations are of special value in the diarrheal cases, the dosage being dependent on their severity. The movements should be reduced to one or, at the most, two a day. Opiates I always avoid, if possible.

Bismuth subnitrate, 10 to 20 grains (0.6–1.3) three or four times a day or more, alone or combined with chalk.

Bismuth salicylate, 5 to 10 grains (0.3–0.6) t. i. d., combined with subnitrate bismuth; or bismuth subcarbonate, 10 to 20 grains (0.6–1.3) t. i. d.; or bismuth subgallate, 5 to 10 grains (0.3–0.6) t. i. d.

Tannigen, tannalbin, or tannopin, 5 to 15 grains (0.3–1.0) t. i. d.

Beta-naphthol-bismuth, 5 grains (0.3) three or four times a day, if there is fermentation. For some of the combinations with kino, catechu, etc., I refer to treatment of Acute Enteritis.

Belladonna can be used for pains, but codein and morphin rarely should be employed.

*Local Treatment.*—This is of great importance, especially when the large intestine is involved, which is usual. The method may be employed by an enema of 1 pint (500 c.c.) to 1 or even 2 quarts (liters) of the solution, preferably employing several quarts (liters), with the recurrent tube or two tubes. If possible I use my own rectal irrigator of hard rubber, or, if the rectum is sensitive, the soft-rubber tube or two catheters.

Normal saline solution—1 dram (4.0) salt to 1 pint (500 c.c.) water—at 105° to 110° F. is excellent if there is much pain. Oil of peppermint (10 drops) can be added to this. Flaxseed tea at the same temperature is useful. Slippery elm solution and gum-arabic solution are excellent soothing applications.

Listerin, glycothymolin, borolyptol, and boric acid, 1 to 2 drams (4.0–8.0) to the quart, are of service.

Tannin, gr. 10 to 20 (0.6–1.3) to the quart (liter); zinc sulphocarbolate, 10 to 15 grains (0.6–1.0); or borax, 1 dram (4.0) to the quart, are good astringents; salicylic acid, 15 grains (1.0) to the quart (liter); irrigation once a day or every other day is of value.

I often employ flaxseed tea or gum-arabic one day, and one of the mild antiseptics or astringents the following day.

In obstinate cases, nitrate of silver solution, 10 to 20 grains (0.6–1.3) to a quart (liter), given once or twice a week, is a valuable adjunct. If the patient complains of pain, a subsequent injection of normal saline solution is of service.

The bowels should be thoroughly emptied, preferably an enema given an hour or two before local treatment.

Protargol or argyrol (1 : 1000 or 1 : 1500) is of service in place of the silver nitrate.

*Surgery.—In cases with evident ulceration,* if no benefit results from medical treatment conscientiously applied for a year, I would advocate appendicostomy or cecostomy with subsequent irrigations. In catarrh without ulcers I do not believe operation is indicated.

## PROCTITIS

The rectum is very liable to bacterial infection, both from within and without, through the anal opening, so that ulcerations and periproctitis may occur. I will briefly refer to this disease, merely to serve as an index to the practitioner.

**Etiology.**—As this condition is often a part of chronic enteritis, the etiologic factors may be identical. Local conditions may also produce it; thus, traumatism, as by a syringe-tip; sodomy; impacted feces; worms; foreign bodies introduced through the rectum or lodged there during their passage from above, such as fish-bones, pins, etc., hemorrhoids, polypi; prolapse, intussusception; tumors; pressure from other organs; displacement of the uterus; stone in the bladder; inflammation of adjacent organs, such as uterus, tubes, prostate, or seminal vesicles. Sitting on cold stones or wet seats may be a cause. Idiosyncrasies to certain foods seem to be a factor.

Proctitis is classified as follows:

1. Acute simple catarrhal proctitis.
2. Chronic proctitis: Atrophic form; hypertrophic form.
3. Specific forms: Gonorrheal, dysenteric, diphtheritic, erysipelatous, and syphilitic proctitis.

**Pathology.**—With simple catarrh there is no pus, except possibly a minute amount. With ulceration there are pus and blood.

The acute and chronic types of intestinal catarrh have already been described. It is preferable to examine pus (if such is present) for gonorrhea, especially if there be a gonorrheal vaginitis, and it may even pass by extension from Bartholin's glands.

Erysipelas may extend from without. Pseudomembranes occur with the diphtheritic type. Amebæ, or the bacilli dysenteriæ, are found with the dysenteric type. We may have the primary chancre of syphilis in the rectum, in which case relaxation of the sphincter will also be noted, or there may be secondary ulceration and catarrh. The test for Wassermann's or Noguchi's reaction should be made if syphilis is suspected. Actinomycosis may rarely occur.

Primary rectal infection from the larvæ of flies (myiasis) has been reported by Nicholson.[1] The patients all suffered from hemorrhoids with rectal prolapse and exposure of the rectal mucosa to fly infection thus resulted. Protection of the prolapsed mucosa prevented subsequent infection.

**Symptoms.**—They have been described. They are: Marked straining and tenesmus; passage of mucus with the stool, or mucus alone with pus and blood if ulceration is present; frequent micturition; throbbing heat and weight in the rectum. Constipation at first, later diarrhea; heavy and aching pain in the rectum and down the limbs; often pruritus and prolapse of the rectal mucous membrane or hemorrhoids; temperature in the acute cases; coated tongue; abscesses of the rectum may develop.

*The chronic cases present less severe symptoms, and no temperature.*

Thus, there may be pain referred to the rectum, or a feeling of weight and pressure within the pelvis or over the sacrum. There may be reflex pain down the left leg, simulating sciatica, dysmenorrhea, dysuria with no discoverable lesions in the genito-urinary tract, swelling of the left leg, anemia, and loss of weight. In young children there are often picking the nose, scratching the anus, straining and protrusion at stool, as symptoms. Mucus, pus, or blood may be found in the feces.

*Digital examination* is painful on account of spasm, and the rectum will be found to be very sensitive and hot to the feel. If the inflammation extend deeper into the tissues, it will feel hard and rigid.

By speculum examination it will be seen to be a deep red, with hemorrhages and erosions. Often the condition is so painful that it is not advisable to use a speculum at first. The chronic cases run a less severe course. Complications: Periproctitis or ischiorectal abscess may occur.

**Treatment.**—Recurrent rectal irrigation with hot normal saline solution at 110° F., or in other cases with cold saline solution at 50°

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[1] Jour. Amer. Med. Assoc., May 21, 1910.

to 70° F., with the patient in the Sims posture for fifteen minutes' duration, is of value, once or twice a day, to relieve inflammation. Flaxseed-tea irrigation is also useful, or the other antiseptics described under Chronic Enteritis.

Injection of hydrastis, 1 to 2 per cent., or aqueous fluidextract of krameria (J. P. Tuttle), 5 to 20 per cent., several quarts, are of service in some cases.

Carbolic acid solutions *should never be employed.*

In the acute conditions I do not care to employ nitrate of silver at first, though later 1 : 2000 every day or two is of value.

Argyrol or protargol (1 : 500 or 1 : 1000) is less irritating. Tuttle suggests the use of the following by injection to quiet irritation after local treatment.

| | | |
|---|---|---|
| ℞. | Flaxseed tea | ℥j (30.0); |
| | Opium | gr. ss to j (0.32–0.065); |
| | Aqueous fl. ext. krameria | ♏xxx (1.77). —M. |

A suppository of opium and iodoform may be substituted.

If there is marked purulent inflammation, then twice daily irrigate with:

Peroxid of hydrogen 8 to 10 per cent., or 1 : 1000 acetozone or alphazone, or even 1 : 10,000 bichlorid of mercury once in twenty-four hours. The latter should be used by the *recurrent method only.*

The bowel should be irrigated well with saline solution after each movement and medicated solutions used once or twice a day.

Injections of starch and laudanum should only be used once or twice a day in severe cases to relieve irritability.

After the acute stage has passed S. Gant recommends spraying the rectum with permanganate of potassium (1 : 3000), or with zinc sulphate, copper sulphate, or nitrate of silver, 1 per cent.

In chronic cases irrigation with nitrate of silver (1 : 2000 to 1 : 4000) is of value, every two or three days.

I have seen an excellent result in a severe chronic case from the injection of the aqueous fluidextract of krameria, suggested by J. P. Tuttle. His formula is as follows:

Macerate 1 pound of bark of krameria in a long percolating tube for twenty-four hours. After this a mixture of glycerin (20 per cent.) and water (80 per cent.) is allowed to percolate through it. The percolate should be constantly stirred and refiltered through the bark a second time.

The filtrate is then evaporated down to 1 pound, thus obtaining an aqueous fluidextract containing gram for gram all the therapeutic properties of the bark. The preparation should be kept in a dark place and not exposed to air.

A 10 to 20 per cent. solution of this can be used for irrigation or a local application of it pure.

The diet and internal medication should be the same as described under Chronic Enteritis. Syphilis, if present, should be treated by "606" or mercury and the iodids.

Warm sitz-baths aid in relieving pain.

## PHLEGMONOUS (PURULENT) ENTERITIS

This disease, a purulent inflammation of the submucous tissue of the intestines, is rare as a primary process. It is probably due to streptococcic infection, the jejunum being most frequently involved. Peritonitis is present, but the purulent enteritis cannot be diagnosed as the cause, in my opinion, until after operation. Phlegmonous enteritis may be secondary to intestinal ulceration, to intussusception, or strangulated hernia. Maragliano has reported septic infection of the ileum *probably by the colon bacillus*, with hemorrhage, ulceration, and peritonitis.

## CHAPTER XXV

# DYSENTERY

Dysentery is defined as an infectious disease characterized by specific ulcerations of the large intestine. In typic acute cases it gives rise to bloody mucous or mucopurulent dejections, accompanied by tenesmus and general symptoms.

Dysentery was known to the ancient world, being first described accurately by Hippocrates 430 B. C., and later by Celsus, Aretaeus, and Galen.

In 1506 the first records of postmortem examinations of dysenteric subjects were published in the posthumous work of Antonio Benevieni. Following these came many writers, among whom I shall mention a few of the most prominent of recent years: Cruveilhier, Virchow, Woodward, Lambl, Loesch, Kartulis, Quincke, Roos, Musser, Osler, Stengel, Stockton, Harris, Councilman, Lafleur, Ogata, Shiga, Russell, Flexner, His, Barker, Duval, Bassett, Vedder, Musgrave, Strong, Craig, and Thomas.

It has been demonstrated that the disease is due to infections of a specific type—either to the amœba dysenteriæ or to the Bacillus dysenteriæ (Shiga) or one of its strains. The disease is transmitted in the same way as is typhoid fever. It sometimes assumes a diphtheritic type, in which case other bacteria are undoubtedly associated.

In the ileocolitis of infants dysenteric bacilli of various strains have been discovered, and some of these cases, both clinically and pathologically, present the appearance of an acute catarrh.

The so-called acute catarrhal dysentery, the sporadic form, I believe, is undoubtedly due to the Bacillus dysenteriæ or one of its strains.

### DIPHTHERITIC DYSENTERY

Diphtheritic dysentery (or, more strictly speaking, *pseudodiphtheritic, as Klebs-Löffler bacillus is not present*) has been shown in many cases to be due to the Bacillus dysenteriæ. *Undoubtedly mixed infections with other bacteria are found in this type.*

They may be found in combination with amebic dysentery, and, in addition, in some of the amebic liver abscesses numerous other bacteria are present, which demonstrate mixed infection from other sources.

Secondary diphtheritic dysentery is a common terminal event in many acute and chronic diseases; and Vedder and Duval have demonstrated that the Bacillus dysenteriæ is present in these cases.

Diphtheritic dysentery in which the diplococcus pneumoniæ has been isolated has been several times reported. In addition, mercurial poisoning or uremia may have this lesion associated. It is evident, therefore, that other bacteria, either present in or entering the intestinal tract under favorable conditions, may produce this lesion.

## PREDISPOSING CAUSES AND CLIMATIC LOCATION

Many causes were formerly given for the production of dysentery, but we may say that they only predispose to infection on account of weakening the organism producing intestinal disturbances. In the case of overcrowding in asylums and camps there is a tendency to unsanitary conditions. By inattention to the proper relation of the latrines, to the water-supply, for example, there may result a severe epidemic.

Dysentery is found in all parts of the world, but is endemic and often epidemic. It is most common in warm climates, such as the southern United States, Cuba, the Philippines, southern coast-line of Asia, Africa, Egypt, Mexico, Central and South America. It has been met with in cold climes, as in North Russia and Greenland; is sporadic in all parts of the United States and occasionally epidemic. Severe epidemics have occurred in the New Zealand States.

During the Civil War, Woodward collected 259,071 cases of acute dysentery and 28,451 of chronic dysentery in the Federal service.

During the recent Spanish-American War the mortality from dysentery and typhoid was far in excess of that from battle; and in the African War the English troops suffered severely. In the Russo-Japanese War the deaths in the Japanese army from dysentery and typhoid were infinitesimal in number, demonstrating that by intelligent care of the water-supply and proper sanitation epidemics of dysentery can be absolutely stamped out.

Dampness, overcrowding, and imperfect ventilation vitiate the system, and so predispose to subsequent infection. With overcrowding and necessarily insufficient and improper attention to sanitary conditions, infection from the dejecta can readily occur, if a sporadic case develop.

Heat and moisture predispose to intestinal disturbances, and readily cause changes in fresh fruits or canned material, if improperly cared for, which in turn produce diarrheal disturbances, and cause susceptibility to infection. Sudden alternations from heat to cold produce the same result. Errors in diet are predisposing causes.

No race or age is exempt from dysentery, and a person going from his native to a warm climate—with the sudden change in food and mode of life incident thereto—is probably more susceptible.

Dysentery is more fatal among the poor and ill nourished than among the rich, though the latter are not exempt. It is probably more prevalent in epidemic and endemic form in small country towns, villages, and farms, where one so frequently sees the well in close proximity to the privy, a stagnant pool, or the family cow-yard.

Dysentery may be endemic, the so-called tropical dysentery; epidemic or diphtheritic; and sporadic (the acute catarrhal dysentery).

It is classified as follows:

1. *Amebic dysentery*, in which there is at times a mixed infection (diphtheritic process).

2. *Bacillary dysentery* (Shiga), or one of its strains, under which are included the sporadic type (acute catarrhal), which probably belongs to this classification, as a catarrhal type (ileocolitis) exists in infants.

The *diphtheritic type* is included and also the *secondary diphtheritic type*, which may be a terminal event in acute and chronic disease, and in which Vedder and Duval have demonstrated the presence of the Bacillus dysenteriæ.

Undoubtedly other varieties of bacteria play a part in the diphtheritic type.

## AMEBIC DYSENTERY

(*Synonym.*—Intestinal Amebiosis.)

**Definition.**—A colitis, latent, subacute, acute, or chronic, caused by the amœba dysenteriæ. There is a special liability to formation of abscess of the liver.

Often these cases occur without the clinical symptoms of dysentery at all. I agree with Musgrave that the condition should be more correctly given the name of " intestinal amebiosis."

The disease is widely prevalent in Egypt, India, the Philippines, West Indies, Southern States, and in tropical countries. It occurs frequently in the United States, and much more in many of our cities than is generally supposed. I have recently attended a case clearly infected in New York State, the patient having never been south. J. P. Tuttle has reported similar cases, as have H. S. Patterson,[1] Stockton, Graser, Braunan, Harvey,[2] and Carey. It is endemic, especially in warm climates, and often becomes epidemic. Sporadic cases occur in temperate climates.

In Manila, Strong states that out of 1328 cases in the United States army, 561 were of the amebic type.

At the Johns Hopkins Hospital, Osler reports most of the acute and chronic cases of dysentery were of the amebic variety; during the first fourteen years there were 119 cases admitted, 95 of which came from Maryland—108 males and 11 females.

**Source of Infection.**—Chiefly from contaminated water or green vegetables and fruit. Musgrave has found the ameba in ice-cream and water-ices.

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[1] Med. Rec., May 14, 1910.

[2] N. Y. Med. Jour., Jan. 21, 1911.

Allen[1] reports 2 cases of apparent contact infection in amebiosis, probably from the ingestion of dried encysted amebæ. These occurred in a tenement, the mother nursing a case, and preparing all food for the family, with subsequent development in other children.

**Amœba Dysenteriæ.**—Lambl first described amebæ in the stools in 1859, and in 1875 Loësch investigated the stools of a dysenteric patient and described the amebæ. He injected the stools into the intestines of dogs and produced ulceration.

Osler, Councilman, Lafleur, Dock, Quincke, Roos, Musgrave, Strong, and many others have carried on investigations.

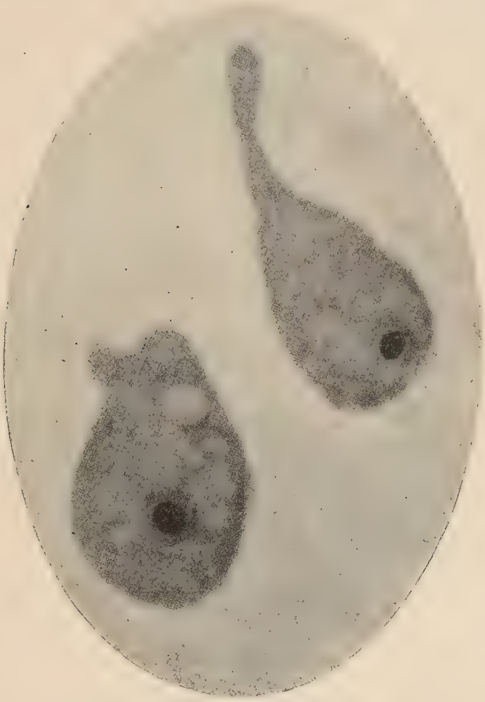

Fig. 239.—Amebæ from a culture. Impression preparation. Borrel's stain (Woolley and Musgrave).

To obtain a specimen for examination little flakes of mucus or pus should be selected, or the mucus may be secured by passing a soft catheter, or through a speculum. Preferably a saline cathartic should be administered, as suggested by Musgrave, and the fluid portion of the stool examined *while warm*. Swollen, altered epithelial cells must be distinguished from the amebæ. The cells are round with granular protoplasm.

*Amœba dysenteriæ* is from fifteen to twenty microns in diameter, and consists of a clear outer zone, or ectosarc, and a granular inner zone (endosarc), and contains a nucleus and one or two vacuoles (Fig. 239). The movements are similar to an ordinary ameba, consisting of a slight protrusion of the protoplasm. They vary somewhat and can be intensified by having the slide heated. They have a pale green appearance under the microscope. Red blood-corpuscles are at times contained in the amebæ, and occasionally bacteria.

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[1] Med. Rec., Jan. 8, 1910.

Musgrave recommends Borrel's stain for the study of amebæ in the tissues.

They may be in large numbers in the tissues. In the pus of a liver abscess amebæ may be abundant. In the sputum from a pulmonary infection from an hepatic abscess they can be recognized.

Quincke and Roos describe three varieties of amebæ in the stools of healthy persons, and Strong two types, only one of which is pathogenic. Allen[1] holds that amebæ are frequently found in the stools of pellagrins, and that they may be of a pathogenic or non-pathogenic variety; amebiosis may complicate pellagra or the diagnosis may be confused. Exclusion of amebiosis is necessary.

Musgrave and Clegg do not think it has been proved there are amebæ non-pathogenic to man. They hold that all such are, or may become, pathogenic. Craig[2] believes the types can be differentiated and Schaudinn classifies two varieties.

Amœba dysenteriæ can be grown in cultures from stools or intestinal ulcers, but not alone as a pure culture. A symbiotic organism is necessary for its development. It has been isolated as a pure culture, in combination with a pure culture of another organism. Fivea, Celli, and Miller claim to have grown it pure, and that it multiplies by division.

Resistant forms of the ameba have been described by Cunningham and Quincke. They are apparently analogous to the gamete forms of the malarial parasite.

The "encysted amebæ" seem, under certain conditions, to be necessary for the transmission of the disease from one person to another, and are regarded by Musgrave and Clegg as the most dangerous type. The free unprotected amebæ, when swallowed, are apparently destroyed by the digestive juices, while in the encysted condition they pass through the stomach and upper intestine unharmed, and in the large intestine grow free and produce dysentery.[3]

Cultures of amebæ have withstood drying for fifteen months.

**Location of Lesions.**—The lesions are found in the large intestine, rarely in the lower end of the ileum; and abscess of the liver is

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[1] N. Y. Med. Jour., Dec. 18, 1909.

[2] Jour. Infec. Dis., June 4, 1908, v, 324.

[3] Marshall, Jour. Amer. Med. Assoc., April 23, 1910.

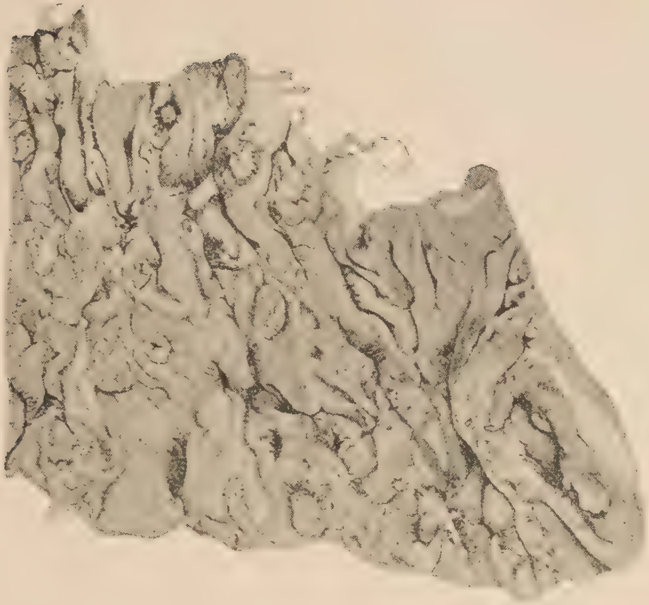

Fig. 240.—Intestinal amebiosis. Cecum. Shows all stages of ulceration. The smallest black points indicate the positions of pre-ulcerative lesions (Woolley and Musgrave).

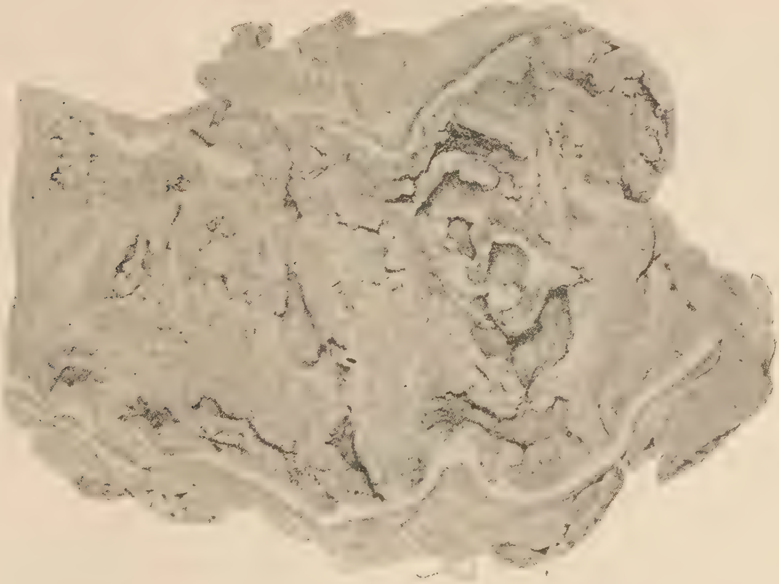

Fig. 241.—Intestinal amebiosis. Rectum. Extensive ulceration and diphtheritis. Thick-walled gut (Woolley and Musgrave).

a common accompaniment, being present in 22 per cent. of Osler's cases.

**Pathology of Amebic Dysentery.**—*Intestines.*—Though writers refer to the classic undermined type of ulcer in amebic dysentery, three types of lesions are described, which may shade gradually into each other:

1. *Pre-ulceration.*—This stage is characterized by the presence of the "small red dots" of Rogers, varying from 0.2 to 0.5 mm. in diameter, and which are intensely congested (Fig. 240). They consist of capillary hemorrhages into the intraglandular tissue. Erosion of the superficial layers of mucous membrane is usually associated.

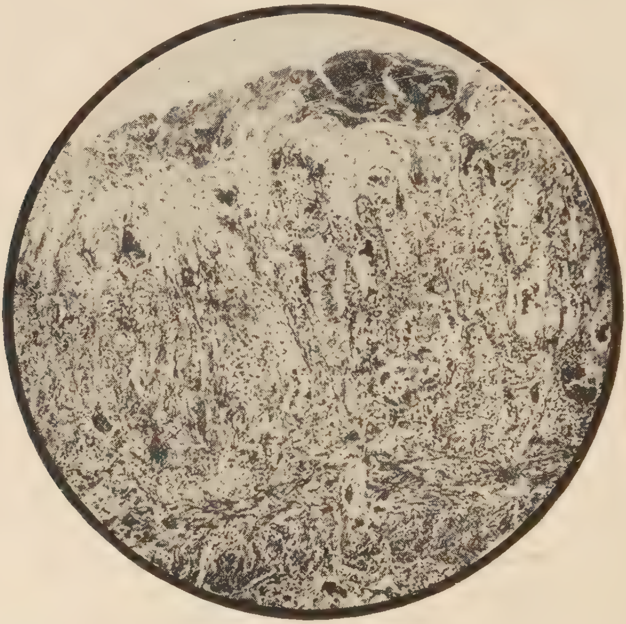

Fig. 242.—Early intestinal lesion. Shows superficial necrosis, glandular distortion, and round-cell infiltration. Borrel's stain. Zeiss obj. A, A, oc. comp. 4; bellows at 30 cm. (Woolley and Musgrave).

There is moderate injection of the mucous membrane and but little thickening of the submucosa. These lesions may be seen in any part of the affected gut, and chiefly in the acute cases.

2. *Ulceration* (*type of Harris*), rarer than the classic type, and believed to be intermediate between the petechiæ and "*undermined*" ulcer. They are probably the result of the superficial erosions, and are primarily confined to the mucous membrane, though they reach into the submucosa or to the circular muscle, but no deeper (Fig. 242). They spread laterally. The ulcer has a punched-out appearance and is round or oval, the edges thick and congested.

Base is clean, gray, and edematous. They often lie at the apex of the intestinal fold and tend to increase in the direction of the short axis of the bowel, and are found in all regions, but less often in the advanced or chronic cases. They are most common in the ileum.

3. *Classic or Undermined Ulcers.*—In the early stage they appear as minute yellow or gray spots in the mucosa, at times at the centers of "Rogers' red dots," and are usually surrounded by a congested area. These spots are the mouths of passages leading to cavities in the submucosa; the mouths and cavities are filled with necrotic material.

As the ulcerative process extends, the cavity in the submucosa is enlarged, and though the necrobiosis eventually involves all the coats, the muscular layers and mucosa are affected less rapidly, and the *latter may be markedly undermined.* The ulcers may gradually coalesce on the surface, or quite frequently the cavities in the submucosa may communicate with each other by tunnels, while the mucous membrane shows a catarrhal condition. The submucosa becomes thickened and edematous, as do often the muscular layers and peritoneal coat (Fig. 243).

The ulcers may be from the size of a pin-head to the palm of the hand. In extensive ulceration the muscular layer may become necrosed or even perforated, and the base of the ulcer be formed by peritoneum or omentum. The omentum plays an *important protective part*, being frequently, early in the ulcerative stage, found adherent to the surface of the intestines. Localized suppuration is, therefore, common. Ulcers may perforate into the subperitoneal or retroperitoneal tissue. They are usually circumscribed, though they may burrow (Fig. 244).

*Healing Process.*—In the case of the small ulcers there may be complete repair, the epithelium from the mucous membrane lining the ulcer, except its base. In larger ulcers there may be considerable scar tissue, which may lead to contraction. J. Cantlie,[1] in the Journal of Tropical Medicine, suggests that amebic infection is probably much more common than is usually supposed. It is known that quite extensive infection can exist without symptoms. He believes that some cases of stricture, or cancer of the sigmoid and rectum especially may be postdysenteric conditions.

According to Musgrave's observation there is established in cases of long duration not systematically treated a chronic *catarrhal condition* with subsequent atrophy (enteritis chronica atrophicans), a thinness of the bowel, absence of normal folds, atrophy of the mucosa, and increased length; and he places it in the classification of sprue or psilosis, believing untreated amebiosis to be one of the causes of this condition. There may be, on the other hand, in some cases localized hypertrophy with well-developed polypi.

*Location of the Lesions.*—Harris believes that in fully one-half the cases the lesions do not extend beyond the beginning of the transverse colon; while Rogers holds that they are more frequently limited to the cecum and ascending colon. The ileum just above the ileocecal valve is *rarely involved*, and generally by only a few ulcers (superficial) and a slight involvement may occur if diphtheritic inflammation complicates. The lower rectum is rarely involved.

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[1] Amer. Med., March, 1910.

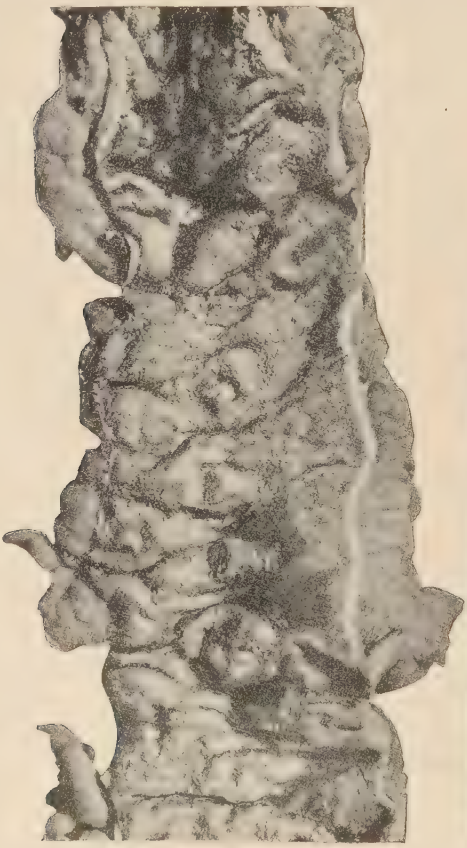

Fig. 243.—Colon. A moderately thickened gut with various types of ulcers (Woolley and Musgrave).

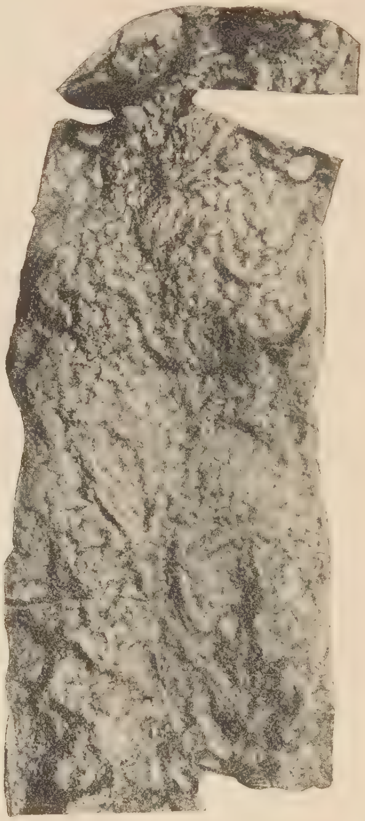

Fig. 244.—Colon. Thin-walled gut, with shallow ulcers, some slightly undermined, others punched out (Woolley and Musgrave).

Strong and Musgrave, comparing 200 cases treated and untreated, find 159 cases involve the entire large bowel, excepting the lower rectum; 23, the cecum and ascending colon; 2, the transverse colon; the descending colon, sigmoid, and rectum, 9; unrecorded, 7; appendix ulcerated, 2 (amebic); appendix involved with large intestine, 14; ileum with large intestine, 7.

The presence or absence of irrigation treatment makes a difference. In cases dying early from intermittent disease, ulceration in the cecum and ascending colon, 11; in descending colon, sigmoid, and rectum, 8; in the entire bowel, 6.

*Microscopic.*—Mucous membrane between the ulcers is little changed. In the neighborhood of the lesions a tendency to *hypertrophy with mucoid degeneration and cyst formation.* In the early lesions, congestion with capillary hemorrhages and edema of the mucosa; increase of lymphoid cells in the interglandular tissue.

Amebæ from 4 to 35 $\mu$ long are present in the glands, interglandular tissues and blood-vessels, muscularis mucosa, and in the veins of the submucoa (Fig. 245).

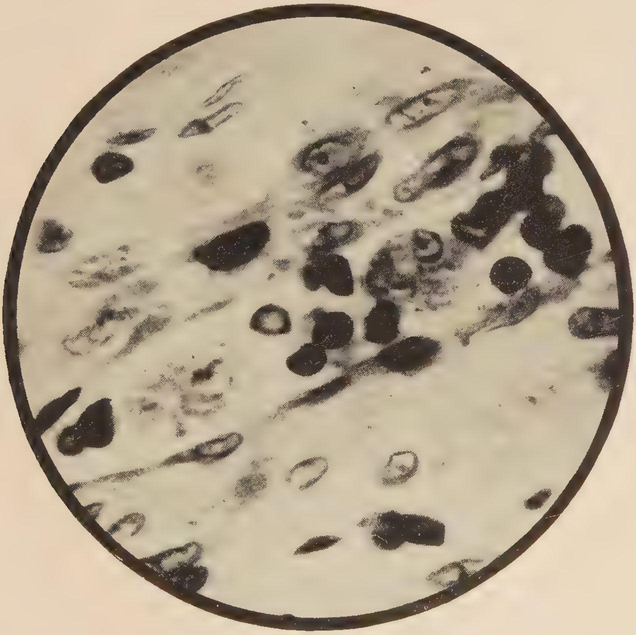

Fig. 245.—Amebæ in a blood-vessel; ×500. Heidenhain iron hematoxylin (Woolley and Musgrave).

In the more advanced condition there is necrobiosis (coagulation necrosis) in the ulcers, with lymphoid infiltration, congestion and thrombosis, and very little polymorpholeukocytic invasion. The amebæ secretion and thrombosis both tend to produce the necrobiosis.

If there is marked polymorphonuclear infiltration, pathogenic bacteria are playing an active part in the process; noticeably then there is diphtheritic inflammation (Fig. 241) or gangrene. When amebæ are found in exudates rich in bacteria, they show evidences of active phagocytosis. In some cases there are many bacteria, but they are probably non-pathogenic, as marked leukocytosis was not present and no process could be seen to be attributed to them.

"In effect, intestinal amebiosis may be said to be rather a subacute chronic inflammatory process, as was demonstrated by the character of the exudate and infiltration, by the early formation of granulation tissue, and by the absence of leukocytic infiltration, a notable absence of purulent inflammation. This applies to the cases not complicated by diphtheritic process or gangrene" (Musgrave).

If the diphtheritic process is associated, some strain of the bacillary type is probably responsible; and if gangrene, some *other bacteria* (mixed infection).

*Lesions (Abscesses) of the Liver.*—There may be local necroses of the parenchyma scattered throughout this organ due to probable chemic products of the amebæ, and also abscesses.

This is quite a common complication of amebic dysentery, and occurred in 27 out of Osler's 119 cases.

Most of the so-called amebic liver abscesses are really local necroses and no pus at all. They may be single or multiple; when single, the right lobe is most commonly affected on the convex surface near the attachment to the diaphragm, or on the concave surface near the bowel.

Multiple abscesses (small and superficial), miliary, containing amebæ, may be scattered throughout the liver substance (Osler).

The hepatic abscess, though it often occurs within the first two to three months after the onset of the dysentery, in some cases may not appear for several years. Some cases are reported as occurring when no dysenteric symptoms had apparently been complained of by the patient.

In the early stage the abscesses are a grayish-yellow color, sharply defined in shape, and contain a spongy necrotic material, with more or less glairy semitransparent fluid in the interstices. The larger abscesses have ragged necrotic walls and contain a viscid greenish-yellow or reddish-yellow purulent appearing material, mixed with blood and shreds of liver tissue.

Old abscesses of a chronic type have a dense fibrous wall. The outer zone is hyperemic; the midzone shows proliferations of connective-tissue cells, compression and atrophy of the liver cells, and an inner necrotic zone. There is the same absence of true purulent inflammation as in the intestines, except where there is a secondary infection with pyogenic organisms. When the latter are present we have true purulent accumulations.

The contents of the necrotic type of abscess show fatty and glandular detritus, necrotic hepatic cells, amebæ, and occasionally Charcot-Leyden crystals. Amebæ are also found in the abscess walls. Cultures are frequently sterile.

Micrococci and bacilli may be found, notably the Staphylococcus aureus, Streptococcus pyogenes, Bacillus coli (with other organisms), Bacillus pyocyaneus, and Micrococcus lanceolatus.

The abscess most frequently points upward and ruptures into the right lung. In some cases an empyema may be produced or a pyopneumothorax. Perforation may occur in other directions, into the pericardium, peritoneum, stomach, intestines, portal or hepatic veins, inferior vena cava, kidney, or externally.

**Symptoms.**—There has been a tendency to arbitrarily divide amebic dysentery into two clinical types, the acute and the chronic, and to create the impression that in acute and chronic dysentery we have necessarily diarrhea or diarrhea alternating with constipation, the passage of blood and mucus, and the presence of tenesmus. Unquestionably, latent and masked infections, with intestinal amebiosis are *by no means rare*, and marked pathologic changes may be present *without objective clinical symptoms*.

Councilman, Lafleur, Osler, Dock, Strong, and Musgrave have reported such cases. The latter notably refers to one case, treated six months for constipation, in whom autopsy showed perforation of a liver abscess as the cause of death, with associated lesions (amebic ulceration) of the cecum and ascending colon.

The early recognition of these irregular types is of great importance, especially when amebic dysentery is endemic. Unquestionably, some of our old cases of apparently simple catarrhal colitis are of this type. In fact, I have had one experience with such a case, where amebæ were found present, with ultimate recovery under appropriate treatment.

Since many of the cases may present no clinical symptoms of amebic dysentery, I believe Musgrave's[1] classification, under "Intestinal Amebiosis," to be the most scientific, which I have but slightly modified.

1. Latent and masked infections with the amebæ.
2. Mild and moderately severe infections (subacute dysentery).
3. Severe infection, including gangrenous and diphtheritic types (acute dysentery).
4. Chronic dysentery.
5. Infection in children and in the aged.

*Latent Infection.*—In these cases there is a pathologic amebic process in the intestines, without any diarrhea or any other symptoms that would indicate the infection.

Musgrave has demonstrated that the symptoms pointing to dysentery may be absent for a long time, yet there are certain symptoms which suggest the condition.

*Dull, aching abdominal pains* are present, which are atrributed to catching cold. They first appear and are most active during the night or early morning. Indigestion, headache, lassitude, coated tongue, and a foul breath are present. There are loss of appetite, some loss of weight, loss of color, and at times a yellow skin.

Physical examination discloses on deep palpation tenderness along the colon, especially *over the cecum and ascending colon*. This last is *a significant symptom;* occasionally thickened intestines can be made out. If a hydragogue cathartic be administered, there will be present in the stool amebæ, mucus, tissue elements, and often old blood.

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[1] Jour. of Amer. Med. Assoc., Sept. 16, 1905.

Musgrave has demonstrated by autopsy on his fatal latent cases that the lesions are in the cecum and ascending colon. Any patient, therefore, living where amebic dysentery is endemic, suffering from the symptoms referred to, should be given a saline, and the stool carefully searched for amebæ. The possibility of amebic infection in chronic colitis with no diarrhea should be considered in New York. I have already referred to a patient infected in that State. On the other hand, some of the latent cases, if untreated, may later develop the symptoms of amebic dysentery.

Under the same class of cases (latent) in regions where amebiosis is endemic we may have patients in whom other symptoms, such as chronic constipation, gastric symptoms, or even appendicitis, may mask the intestinal amebiosis. Musgrave has reported such cases.

*Mild and Moderately Severe Cases (Subacute Dysentery).—Mild Cases.*—These frequently develop from the latent type, and often present the aspects of a *diarrhea* and not of dysentery. Abdominal pain, tenderness along the colon, headache, digestive disturbances, irritability, melancholic condition, anemia, and loss of weight are present.

Amebæ are found in the diarrheal movements. Some of these cases never show dysenteric movements, even without treatment. This type Musgrave believes often becomes chronic; while, on the other hand, chronic gastro-enteritis or " sprue " may be the ultimate outcome, giving their clinical pictures. The patient usually dies from intermittent disease or complications.

*Moderately Severe Cases (Subacute Dysentery).*—In these cases with the symptoms just described there may be a diarrhea more marked in the morning, consisting of several semifluid stools, no mucus or blood, and passed without pain. This may intermit with constipation. Finally, the attack may increase in intensity, and mucus and blood will appear. In other cases they will occur from the onset. Usually the more acute the onset, the more rapidly the severe symptoms develop.

*Severe Cases (Acute Dysentery).*—These are the classical cases usually described. Diphtheritic and gangrenous processes, due to some secondary infection, are most common in this type.

The onset is usually quite sudden, whether primary or following on a milder type. There are marked abdominal colic; diarrhea; tenesmus, which may be constant and very painful; straining, and then, finally, passages chiefly of small quantities of mucus and blood. In some cases the dejecta are hemorrhagic, consisting of pure blood or dark and coagulated blood. Sloughs are passed in others, consisting of gray or blackish masses of necrotic tissue of very foul odor.

The temperature, as a rule, is not high. The patient rapidly emaciates, and the heart becomes rapid and feeble.

Death may occur in severe cases within a week from the onset. Hemorrhage (intestinal) or perforative peritonitis may take place. Many cases recover, but some become chronic. In others extensive ulceration may remain after sloughing and the diarrhea continues, the patient rapidly emaciating, and finally dying exhausted within a couple of months. Emaciation is very marked in these patients. Corneal ulceration may occur.

*Stools* —As many as twenty to thirty may be passed in twenty-four hours, colicky pains usually precede them, and they are followed by straining and severe tenesmus. Movements at first are copious, later scanty, and consist chiefly of mucus and blood. Intermissions and exacerbations of diarrhea occur, gangrenous dejecta, dark red brown and containing gray or black fragments of tissue (foul in odor), may be passed, or pure blood. Amebæ are present in the stools.

*Abdominal Pains.*—These are quite severe, chiefly before evacuation, and sometimes they are continuous. They are frequently located in the umbilical region and left iliac fossa; at times in the right iliac fossa, and may simulate appendicitis. Pressure increases the pain.

*Tenesmus.*—This consists of pressure and constriction in the rectum and a desire to go to stool. It may be continuous and accompanied by dysuria or strangury.

*Other Symptoms.*—Fever may occur, generally of moderate type, and it may be accompanied by chills at the onset. Temperature is irregular. Anorexia, nausea, and vomiting may occur. There may be severe prostration, cold extremities, delirium, stupor, drowsiness, and cerebral disorders.

*Chronic Dysentery.*—This type of dysentery shows several forms. It may be rather mild, characterized chiefly by diarrhea, with no blood or tenesmus, following on the mild type previously described. This may continue a number of years.

Dysentery in more marked cases is subacute from the onset or gradually passes into the chronic stage. There are generally alternating periods of diarrhea and constipation covering several years. During the exacerbations there are pain, passages of blood and mucus, tenesmus, and a slight rise of temperature. Many such cases do not feel especially ill between attacks and keep fairly well nourished. The appetite is often irregular in this type, and errors in diet are followed by exacerbations of the disease. The tongue is red, glazed, and beefy.

In more severe cases emaciation may be extreme and the patient be confined to bed most of the time. There are loss of appetite and nausea; diarrhea is quite persistent, there being mucus or mucus and blood in the stools, with attacks of colic and tenesmus. There may be some periods of improvement.

*Infection in Children and in the Aged.*—Musgrave reports cases in children from the age of six months to ten years in the Philippines. Children seem to present a natural immunity, he believes, and when infection does occur it seems of a mild type and readily yields to treatment.

The symptoms resemble those of the mild type in adults. In the aged there also seems to be a natural immunity, but when the disease was established it ran a severe and rapid course.

Liver abscess is infrequent in the very young and aged; Musgrave notes it is infrequent in the natives.

The only certain method of diagnosis is by microscopic examination of the feces or discharges and finding the amebæ.

Other parasites are found at times associated with the amebæ, such as the trichomomedæ, ova of uncinariæ, embryo strongyloides, tenia, oxyuris, etc.

*Circulatory System.*—The pulse may be of good quality at first; later, rapid and feeble. There are the changes of secondary anemia. Early, the blood is normal; later, the red cells are those of anemia. Still later, they may become irregular in size and shape and the count be reduced.

*Leukocytes.*—An increase in polynuclears and eosinophiles may occur. Hemoglobin decreases with the red cells. Spleen usually not enlarged.

*Temperature* may be absent, moderate, or intermittent. In cases that are complicated, especially if there is diphtheritic inflammation of the colon, it may become quite high. With liver abscess temperature is frequent, but not constant. If there is mixed infection in the liver, bacteria with the amebæ, the temperature may be intermittent or remittent and resemble malaria or endocarditis. Sometimes it may be subnormal.

*Nervous System.*—Neuritis and neuralgias may be present.

*Pain.*—This varies in type and intensity.

*Tenesmus.*—More often it is an indication of secondary involvement or complications. It is never present in the latent forms, and is often absent or very slight in the moderately severe cases.

In acute cases with diphtheritic process or secondary infection, as from Bacillus dysenteriæ (Shiga), it may be severe. It is more likely absent when the lesions are in the cecum or upper colon.

*Colicky pains* frequently occur, and at times severe colic.

The dull, aching *abdominal pain* is often prodromic and persists during the course of the disease, at times interfering with the patient's rest. The greatest intensity is usually along the colon, as demonstrated by palpation. Sometimes it is confined to the cecum. It may be complained of in the back. It is probably due to the ulcerative process in the bowel and—as demonstrated in the latent cases, as shown by postmortem—is the only indication during life of serious lesions. These pains are often the worst at night.

*Burning pains* ("heart-burn in the abdomen"), Musgrave states, may be general or local; when the disease is in the sigmoid or rectum these pains may be intense, and extend down the backs of the thighs to the calves, and old cases of sciatica may be started up. Musgrave holds that the beginning of sciatica, associated with such cases, is of sufficiently frequent occurrence to be suggestive.

Appendicitis pains are at times stimulated, but examination will show that the tenderness is located in the caput coli. The differential method by Morris' point would be of value in these cases, as described under Appendicitis.

Neuralgia, myalgia, and arthralgia may be present. There is persistent dull headache in the back of the head and neck.

The genito-urinary, respiratory, and special senses are rarely involved, as is also true of the joints and osseous systems, though complications may occur.

**Diagnosis.**—In amebic dysentery there are so many clinical types that the diagnosis is by no means easy, and can only be made absolutely by microscopic examination of the feces.

In regions where the disease is endemic and the microscope is not available, one can reasonably infer its presence by the most valuable symptom, to which I have already alluded, namely, "abdominal soreness, which is increased on pressure, and extends along the course of the colon, especially when there is maximum intensity over the cecum and ascending colon."

If bowel movements are present, their odor and the appearance of blood are of chief diagnostic import. Indigestion, pain, nausea, and other symptoms are not as important.

Often a thickened tender colon may be felt on palpation. These symptoms give the nearest approach to diagnosis without microscopic examination of the feces. Loss of weight is a guide to the infection.

Musgrave believes that the presence of amebæ in the stools in tropical regions should be considered diagnostic for purposes of treatment.

Amebiosis should be treated, even if other complications are present.

**Prognosis.**—The disease is generally milder in children and in the natives of the tropics. The course is shorter and the mortality higher among the aged. Previous good health is a favorable factor.

The *shorter the duration of the disease* and the *earlier the local treatment*, the *better is the prognosis*.

The higher up the lesion, the greater the mortality and the less active the clinical symptoms of dysentery. Infections of the cecum are the most serious.

Under proper treatment recovery is the rule in young and well-nourished adults if the disease is not of long duration. The early diagnosis and treatment are the important features, as otherwise the apparently mild cases may assume a dangerous character. Tendency to relapse or chronicity are characteristics. Abscess of the liver is a serious complication.

**Treatment.**—*Medical.*—The disease is contracted probably in the same way as typhoid fever, and the same prophylactic measures should be used. Infection through the drinking-water is undoubtedly the chief method.

Musgrave holds that the best rule ro observe in countries where the disease is endemic is to " take nothing into the gastro-intestinal tract which has not been sterilized." He has found the amebæ in the drinking-water; on dishes washed in tap-water; in the soil from contamination; on the surface of uncooked vegetables, such as lettuce; on raw fruits; from hand contamination; and in ice-cream, water-ices, and milk.

*Prophylaxis,* when the disease is endemic or during epidemics, is very important.

All drinking-water should be boiled, and dishes should be washed in boiled water, also the hands.

Raw fruits and vegetables should first be placed on ice, and then have scalding water poured over them, which kills the amebæ. Ice-cream and water-ices should not be taken.

The vaginal douche and, especially, rectal enemata from tap-water should be avoided.

The stools should be disinfected in carbolic acid (1 : 20) or in bichlorid of mercury (1 : 1000), and the same precautions taken with linen—soaked in carbolic acid (1 : 20) and boiled.

Care of the hands and the prevention of fly infection (by screens) are necessary.

We have already noted that mixed infection with the Bacillus dysenteriæ (Shiga) may be present.

The *acid of the stomach lessens the chance of infection,* and *acid mixtures* may be given, such as *dilute hydrochloric acid.*

In *acute dysentery* the patient should be put to bed and placed on a liquid diet: barley-water, rice-water, bouillon, broths, gruels, white of raw egg, tea, also peptonized milk diluted with lime-water (at least 25 per cent. or more), or peptonized and diluted, or equal parts of milk and barley-water. With some sour milks agree, as koumiss, etc. Turnip-top purée has been advocated as of value.

*Sanatogen* is an excellent preparation and somatose is of service. Personally, I prefer milk-free diet and only the strained broths, gruels, etc., and 10 per cent. gelatin solution flavored with vanilla, 4 to 6 ounces (125–185 c.c.). Give nourishment in divided doses (2 quarts daily) and a large amount of acidulated water. If the temperature is over 103° F., only water is given until defervescence to that point. Later the diet can be increased.

Hot applications or poultices should be employed over the abdomen for the relief of pain or colic.

*Internal Medication.*—Musgrave and Osler both object to the use of bismuth preparations for the diarrhea, on the ground that they coat the ulcers and interfere with their local treatment. I believe this probably to be true as regards bismuth subnitrate or subcarbonate, which would be required in large doses. I have occasionally employed bismuth subgallate, 5 to 10 grains (0.3–0.6) t. i. d., in combination with other remedies, apparently with benefit.

On the other hand, I have seen at times that, in spite of all treatment, too frequent movements continue either in the acute or chronic cases. In such event I have employed bismuth subnitrate, as much as 90 to 120 grains (6.0–8.0) in divided doses in twenty-four hours, with good results. This is preferable to the use of opiates, and I only employ it to avoid such. I have never had poisoning from large doses of bismuth subnitrate. It would seem a more likely occurrence in children.

At the Ancon Hospital, Deeks and Shaw employ chiefly the subnitrate of bismuth treatment, 1 to 1½ drams (4.0–6.0), by measurement, or 3 drams (12.0), by weight, stirred in a glass of water, every three hours until the patient improves, which may be from three to fifteen days. Normal saline or plain water irrigation of the bowels are added. Rest in bed and an absolute milk-diet are enjoined. An occasional dose of morphin and atropin may be required to relieve tenesmus. They claim fewer fatalities by this method than by other courses of treatment. Finally, if improvement does not follow this treatment, and the acute symptoms are severe and toxic, they recommend surgical interference (appendicostomy or cecostomy), as advocated by Herrick. The latter suggests this treatment for the following conditions: First, appendicostomy in chronic cases; second, appendicostomy or, preferably, cecostomy in acute fulminating and toxic cases. By means of cecostomy, especially through the temporary artificial anus, rest is given to the diseased bowel, and it also can be kept irrigated. For the literature, the reader is referred to the Medical Record, Nov. 13, 1909. The writer hardly advocates an artificial anus in acute cases, and doubts the advisability of the bismuth treatment, except as previously noted.

In the initial stage the patient should be given magnesium or sodium sulphate, 1 to 2 drams (4.0–8.0), one or two doses, so as to thoroughly cleanse the bowels. Calomel, 5 grains (0.3), may be given, or a single dose of castor oil, 1½ ounces (45.0).

In young persons these remedies should be administered in smaller doses.

Some recommend 20 minims (1.18) of laudanum, followed in half an hour by 20 to 30 grains (1.3–2.0) of pulv. ipecac, after the saline treatment; but, like Musgrave, I believe this of no special service in these cases.

Brem and Zeiler[1] report excellent results at the Colon Hospital in the treatment of intestinal amebiosis from the use of ipecac pills freshly coated with fused salol $\frac{1}{16}$ inch thick. By this means the ipecac passes through the stomach unattacked by the gastric juice; vomiting is prevented and no opiate is necessary. Simon[1] has used a salol coating $\frac{1}{8}$ inch thick, but many pills passed through the intestines undissolved. They found the best method usually was to begin with 60 to 80 grains at bedtime, 8 to 9 P. M., decrease the dose by 5 grains daily until a dose of 10 grains (in all) is reached, and then, as a rule, stop. Occasionally rapid cures may be effected by giving 40 grains t. i. d. These writers observed that milk curds or solid food delayed the passage of the pills until the salol coat was corroded, and vomiting ensued. They, therefore, direct that *no solid food* or milk should be given for at least six hours previous to the ipecac, and no liquids for three hours previous. No opiate is necessary. Have the patient lie on the right side to aid passage of pills from the stomach. The patient should remain in bed and the general diet should be liquid. Enemata of normal saline solution, quinin, thymol, and thymol and quinin combined, 1 : 500 to 1 : 1000 each, are useful.

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[1] Amer. Jour. Med. Sci., Nov., 1910.

Dock[2] has reported excellent results from the ipecac treatment. Leonard Rogers[3] reports favorable results in Calcutta in the prevention of tropical abscess (amebic) of the liver by the treatment of the presuppurative stage with ipecac. He suggests the use of capsules of ipecac keratin coated. A shellac coating can also be employed (author), while Sandwith advocates capsules of animal membrane. Anders[4] starts the treatment with calomel (several doses), and then administers magnesium sulphate, 1 dram (4.0) every third hour, until all hardened fecal masses are removed. Saline laxatives, however, should not be continued after the dysenteric have been converted into diarrheal dejecta. Anders advocates also the use of ipecac administered by salol-covered pills in the earlier stages of the disease, not less than 30 grains (2.0) of ipecac on the first day, and diminishing the dose daily by 5 grains (0.3), so that by the sixth day only 5 grains (0.3) are administered, and for a week or ten days this nightly dose of 5 grains (0.3) should be continued. He adds to the treatment daily high enemata of quinin solution, 1 : 5000, gradually increasing the strength to 1 : 1000. In obstinate chronic cases appendicostomy with bowel irrigation is advocated. The ipecac treatment has always seemed to the author purely empirical, and the question has always arisen in the writer's mind as to how many *permanent cures* result from ipecac treatment.

Occasionally, salol, 3 grains (0.194), with guaiacol carbonate, 3 grains (0.194), and 1 grain (0.065) of pulverized ipecac, given several (three or four) times a day, with small doses of Dover's powder, 2 to 3 grains (0.13–0.194), have proved of service according to some reports. Kho-sam (Brucea Sumatra or Brucea antidysenterica), in 1-grain pills three or four times daily, has been suggested in place of ipecac by Lemoine[1] and Schneider.[2]

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[1] Jour. Amer. Med. Assoc., 1919, liii, 1526.

[2] N. Y. Med. Jour., July 10, 1909; ibid., Dec. 4, 1909.

[3] Therap. Gaz., June 15, 1909.

[4] Jour. Amer. Med. Assoc., Feb. 12, 1910.

Strong has reported good results in some cases by giving internally acetozone (1 : 5000 or 1 : 3000) in carbonated water, 1 to 2 liters (quarts) in twenty-four hours in divided doses.

I have employed 1 liter (quart) of acetozone (1 : 1000), given in divided doses by mouth during the day with good results. Just before administering, each dose can be flavored with orange-juice to make it more palatable.

Among valuable astringent remedies are:

Salicylate of guaiacol (guaiacol-salol), 5 to 10 grains (0.3–0.6); tannalbin, 10 grains (0.6); tannigen, 10 grains (0.6); or tannoguaiaform, tannopin, and tannocol, given in doses of 5 to 10 grains (0.3–0.6) three or four times a day.

Hydrochloric acid with pepsin or alone, or nitromuriatic acid are of value. Musgrave recommends hydrochloric acid. Thus:

℞. Acidi hydrochlor. dil. }
Comp. tinct. cinchona } ................. āā ʒiij (12.0);
Aq. destil. ........................... q. s. ℥iv (125.0).—M.
Sig.—ʒj to ij (4.0–8.0) in water t. i. d. before food.

Vomiting should be treated by the methods described under Acute Gastritis. Small doses of Dover's powder may be required for persistent diarrhea.

*Local Treatment.*—This is of *extreme importance*, not only in the acute, but also in the latent and chronic cases.

Extensive researches were conducted by J. B. Thomas, reported in Bulletin 32, Bureau Government Laboratory, Manila, who found the following solutions destructive to amebæ or inhibiting their growth:

Acetozone, 1 : 1000, most destructive to amebæ, and alphozone, 1 : 1000, nearly as much so.

They destroy other bacteria as well.

Protargol and argyrol, 1 : 500, were excellent antiseptics. Sulphate of quinin, 1 : 500, preferable strength, or bisulph-quinin; nitrate silver, 1 : 2000, of service; thymol, 1 : 2500, readily destroys amebæ, also permanganate of potash, 1 : 2000, is useful.

Hydrogen peroxid was recommended by Harris some years ago, and I have used it successfully for some time.

Cold water (under a temperature of 45° F.) has been highly advocated by J. P. Tuttle as destructive to the amebæ, and also removing them from the bowel. He sometimes employs 5 to 10 per cent. hydrogen peroxid in this injection, and places the patients in the knee-chest position and has them retain the injection for a considerable time (one-half hour).

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[1] Bull. Path. Exot., Paris, 1908, i, 72.

[2] Ibid.

A *glass irrigator*, attached to a colon-tube, with the opening preferably at the end, can be employed; if the ulcers extend low down into the rectum or there is extreme tenesmus, then an ordinary rectal tip.

The foot of the bed should be elevated 12 to 18 inches and the patient placed in the Sims position, or the hips can be elevated on a pan. The patient's position should be changed, and he should be moved so the fluid will gravitate into the caput coli by movements described as "rotation method" under Enteroclysis.

Musgrave recommends the injection of at least 1 to 2 liters (quarts), which should be retained five to fifteen minutes, preferably the latter. At the commencement, if there is much irritation, often a smaller quantity must be used.

The knee-chest position, advised by Tuttle, is excellent in the latent or chronic cases, but I would not advise it in acute conditions.

If there is much irritation, one can precede the antiseptic injection one-half hour by a small enema of normal saline solution—2 ounces (60.0) containing $\frac{1}{4}$ grain (0.016) of morphin—or with tincture of belladonna, 10 minims (0.592), alone or together.

This should only be done once or twice in the first twenty-four hours and not repeated.

Musgrave has suggested taking advantage of the action of reversed peristalsis by giving occasionally a preliminary enema of a 7 to 8 per cent. salt solution, about 1 pint (500 c.c.), containing $\frac{1}{4}$ grain (0.016) of morphin, one-half hour before the antiseptic injection.

He then employs acetozone (1 : 5000 to 1 : 3000), combined with quinin (1 : 1000 to 1 : 750), or gives the injections alternately, employing one to five enemata every twenty-four hours, according to the severity of the case.

Alphozone, same strength, or hydrogen peroxid (1 : 10) can be substituted.

We must remember that quinin affects some cases badly, causing gastric symptoms, vomiting, and headache. It must be omitted or weaker solutions given to such patients.

Quinin is much more efficacious in the strong solutions (1 : 1000 to even 1 : 500). The usual solutions suggested are much too weak. One can take advantage at the same time of the cold injections, as suggested by Tuttle, unless the patient have an idiosyncrasy to cold or renal complications. In such event a hot injection at 120° F. is of service.

An excellent method is to alternate, giving an enema of acetozone (1 : 1000), and the next enema of quinin (1 : 1000 to 1 : 500), cold, at 50° to 40° F.

If the quinin disagrees, then alternate the acetozone or alphozone with thymol (1:2500), or protargol or argyrol (1:500). The silver nitrate is at times found irritating in the acute cases.

Permanganate of potash (1:2000) is also of service, or hydrogen peroxid, 4 ounces (125.0 c.c.) to 1 liter (quart).

The local treatment is of chief importance to check the lesions and prevent liver abscess. In the latent cases the bowels should be opened freely with magnesium sulphate and daily injections of quinin and acetozone given. In cases of marked tenesmus, where the large or even small injections cannot be retained, recurrent irrigations with the antiseptic solutions, 1 or 2 gallons—1 pint (500 c.c.) being kept in the bowels, with marked elevation of the bed and the solution of one-half strength—are of great service.

*Chronic Dysentery.*—In the *chronic cases* the diet should be quite liberal, but indigestible and *rich food* should be avoided. Mashed potatoes, boiled rice, and constipating food are often indicated in the diarrheal cases, and the avoidance of fruits and green vegetables. Milk, eggs (raw), the sour milks, and fats are of value. Judgment must be used in each case as to what will agree, whether liquid, semisolid, solid food, or combinations. Sanatogen is of value, and the iron preparations are often indicated. The tannic acid preparations, as suggested in the acute cases, should be employed. Occasionally large doses of bismuth subnitrate are necessary. Quinin and acetozone injections should always be employed; and nitrate of silver (1:3000 to 1:2000) is of value, used once or twice a week to heal the ulcers. Change of climate is useful.

In all cases of fever, sponging (alcohol) is the best method of treatment. I deprecate the use of antipyretics. If heart stimulants are required, small doses of strychnin, caffein, and spartein, or camphorated oil by hypodermic are indicated.

I have, moreover, a suggestion which I believe may prove of value. Piffard and Tousey have demonstrated that Morton's claims regarding the production of internal fluorescence by the *x*-rays after the administration of small doses of fluorescein or quinin are fallacious. However, the use of $\frac{1}{4}$ grain (0.016) fluorescein in 6 ounces (200 c.c.) of water, and placing the patient in the full electric-light bath for fifteen minutes or more, I believe, might prove of service in latent, chronic, or even in acute dysentery. There is great heat penetration from this light bath, as has been demonstrated in rheumatic and other conditions, and necessarily a certain penetration of light rays. The heat, light, and fluorescence are destructive to amebæ. The fluorescein solution should be given by high enema; soda bicarbonate, 15 grains, should be added before injection, and water, q. s. 1 quart (liter). Musgrave reports to me the disappearance of filaria after the administration of fluorescein and the use of light. He has demonstrated that violet light, *x*-ray light, and fluorescence inhibit amebic action.

*Carbonic Acid Gas for Tenesmus.*—Rose has shown that the injection of $CO_2$ into the rectum will relieve tenesmus. It is worthy of use as an adjunct and can be given by his bottle.

Intestinal hemorrhage from dysenteric ulcers should be treated by high injections of 10 per cent. gelatin or, preferably, Trémolière's solution, or, rather, a modification:

*Trémolière.*—Gelatin, 5 per cent. solution, containing calcium chlorid, 2 per cent.

*Trémolière (Modified).*—Calcium lactate, 20 grains (1.3); gelatin (10 per cent.), 8 ounces (500 c.c.).

Hot (120° F.) or cold (40° F.) astringent injections—1 dram (4.0) alum to 1 pint (500 c.c.)—may be required. Avoid cold if there is shock.

Morphin, $\frac{1}{8}$ to $\frac{1}{4}$ grain (0.008–0.016), by hypodermic at once; ernutin, 5 minims (0.296), hypodermically, or fluidextract of ergot, 1 dram (4.0), internally; ice-bag locally; calcium chlorid or lactate, 10 grains (0.6), by mouth every three hours, gelatin solution (5 per cent.) may also be taken by mouth. Hypodermoclysis may be required if there is marked shock, or even infusion. Horse serum or human serum may be required in severe cases.

*Surgical Treatment.*—In cases of chronic dysentery of long duration which do not respond to medical treatment, or in such cases with repeated acute exacerbations, surgical procedure is indicated. This is true of either the amebic or bacillary type of dysentery.

*Appendicostomy.*—This operation was first suggested by Weir. It consists in suture of the appendix to the abdominal wall and skin, and removal of its apex. Subsequent irrigations of the large intestine are carried out by means of a small tube or catheter, which is inserted through the lumen of the appendix.

*Cecostomy.*—An incision is made over the cecum and the latter brought into the abdominal wound. The cecum is incised and sutured to the abdominal wall. A catheter or drainage-tube is then inserted for the purpose of irrigation of the colon. Gibson has devised a valve operation, separating the abdominal muscles in a special manner, so as to prevent leakage and ultimately secure a more ready healing. Cecostomy is indicated when the appendix is diseased or in an abnormal position, so that appendicostomy is impossible.

*S. G. Gant's Modified Cecostomy.*—Gant[1] makes his incision over the cecum nearer the ileocecal junction. After opening the cecum and suturing it to the abdominal wall by means of a special director, he inserts a drainage-tube or catheter through the ileocecal valve into the ileum. A second tube is inserted into the cecum. In cases where ulceration of the ileum is present, it is thus possible to irrigate both ileum and colon. With amebic dysentery the ileum is rarely involved.

Excellent results have been reported from these methods.

*Abscesses of the Liver.*—These should be opened and drained.

[1] N. Y. Med. Jour., Aug. 15, 1908.

Rogers[1] states that in the presuppurative stage of amebic abscess of the liver there is an exacerbation of temperature, with usually, but sometimes without, pain in the liver region, and leukocytosis is present. He recommends ipecac as a preventive of suppuration. With suppuration there is increased density of the liver shadow to the *x*-rays, local swelling, and edema with increasing leukocytosis. Rogers holds that 86 per cent. of these abscesses are sterile and are infected by other bacteria by the open operation. He recommends repeated aspirations of the abscess-cavity with the injection of quinin solution *without drainage.* If this method fails, add sterile siphon drainage. Ipecac is also given.

## BACILLARY DYSENTERY

**Definition.**—A form of colitis, frequently an ileocolitis, usually of an acute type; occurring sporadically and in severe epidemics; attacking children as well as adults, and characterized by pain, tenesmus, and the frequent passage of blood and mucus; the result of infection by a specific bacillus, of which there are various strains.

**Etiology.**—Owing to improvement in sanitary conditions, bacillary dysentery is less frequent. This is the type which has proved such a scourge, as epidemics in crowded asylums, institutions, and camps. It is one of the great camp diseases, and I have already referred to the fact that Woodward collected 259,071 acute cases during the Civil War. The disease prevails in the Philippines, Porto Rico, Cuba, and in South Africa. In Japan a fatal type has prevailed, especially in summer and autumn, having a mortality of over 25 per cent. In 1899 there were 125,489 cases with 26,709 deaths, collected by Eldridge. Most of the severe epidemics in the tropics are of the bacillary type, and the same form prevails in the temperate climates.

*Bacillus Dysenteriæ.*—In 1892 Ogata, during an epidemic of dysentery in Japan, isolated fine bacilli which, when introduced by the mouth or rectum, produced ulceration in the intestines of cats. In 1897, during a severe epidemic, Shiga isolated the Bacillus dysenteriæ and described its special characteristics, demonstrating it to be the specific cause of the disease. Flexner and Barker found in the dysentery in the Philippines an identical organism, and Strong, Musgrave, and Craig have made a careful study of it.

It has been found in acute dysentery in Porto Rico. Out of 1238 cases of dysentery in Manila, Strong and Musgrave report 71 of the bacillary type, 51 suspected bacillary, and 561 amebic.

Kruse, in Germany, has isolated an identical bacillus. Vedder and Duval demonstrated that sporadic cases in adults in Philadelphia, and also epidemics in the Lancaster County Asylum, Pennsylvania, and in the Almshouse, New Haven, were due to the Bacillus dysenteriæ. Duval and Bassett, during the summer of 1902 at Mount

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[1] Philippine Jour. of Sci., July, 1910.

Wilson Sanitarium, first demonstrated that certain forms of summer diarrhea in infants were due to Bacillus dysenteriæ, and under Flexner's direction at the Rockefeller Institute investigation into the cause of infantile diarrhea in New York, Boston, Philadelphia, and Baltimore, showed the Bacillus dysenteriæ present in 63 per cent. out of 412 cases.

Several strains of this bacillus have been found, and the Flexner-Harris type is the one most frequent in the United States. These strains have been determined by the relative agglutinative power of immune serum upon the bacilli isolated, and also by the action of the bacilli upon various sugars. Flexner recognizes three types:

1. "Shiga type" attacks glucose, without action on other sugars, including mannite and lactose.

2. "Flexner-Harris type" attacks glucose, mannite, and dextrin, not lactose.

3. "Bacillus" (Hiss and Russell) attacks glucose and mannite. No action on dextrin and lactose. His now recognizes four groups based on fermentative characteristics, and Shiga has added a fifth, intermediate between the acid and non-acid bacilli.

The lesions produced by the different strains are identical. The organism agglutinates with the blood-serum of cases with acute dysentery, as well as with the serum of immunized animals. The Flexner-Harris type agglutinates in dilutions of 1:1000 to 1:1500.

In two instances the organism has been isolated by Duval in the stools of healthy children. In *dysenteric stools it is most readily isolated from the particles of mucus.* The organism has not yet been isolated outside the human body, but the belief is that it is probably water-borne, and that the same prophylactic measures should be taken as in typhoid fever. Camp epidemics clearly originate in this way, and the care taken by the Japanese in regard to the latrines and water-supply practically eliminated the disease during their recent war.

Bussow[1] describes a small epidemic of dysentery in a hospital ward due to the cook as a carrier. Flexner bacilli were recovered from the stools. The cook was suffering from diarrhea, but without general symptoms.

**Morbid Anatomy.**—In acute cases, when death has occurred during the first week, the mucous membrane of the large intestine is swollen, hyperemic, of a deep red color, and presents elevated coarse ridges and folds. There are ecchymotic patches scattered throughout the swollen mucosa; over the surface there is usually a superficial necrotic layer, which can be brushed off lightly with the finger. This may be in patches or over large areas. There is no ulceration, but only the superficial general necrosis of the mucosa. They are, in effect, superficial erosions which give it a worm-eaten appearance. This superficial necrotic layer is, in effect, a fine pseudomembrane.

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[1] Münchener medizinische Wochens., Dec. 27, 1910.

The solitary follicles are swollen and red, but their prominence is obscured in the involvement of the mucosa.

In severe cases the entire coats of the colon may be stiff and thick, and the mucous membrane greatly increased in thickness, grayish-black in color, extensively necrotic, and in places gangrenous. The submucosa is often enormously thickened and edematous. The serous surface is often deeply injected, and the vessels of the mesentery, especially near the sigmoid and rectum, may be distended.

The ileum for 10 or 15 cm. is quite frequently involved (which is rare in amebic dysentery), having a deeply hemorrhagic mucosa with superficial necrosis. Peyer's patches and the solitary glands may be moderately swollen. The specific bacilli and various cocci are abundant in the necrotic mucous membrane, and are said by Strong to be seen in all the coats. Streptococci and various other types may be present.

In the subacute cases there is less thickening of the intestinal walls, there is less necrosis, the solitary follicles are more swollen, the mucosa less red, there are superficial erosions, and no ulcers. The disease, as suggested by the complications, is evidently characterized by a more or less acute *general toxemia*, starting from a localized process. The toxins of dysentery are probably excreted by the bile and also through the intestinal mucosa, thus aiding in the damage to the intestines. Probably the toxins are responsible for cerebrospinal lesions (Herter).

**Symptoms.**—The incubation period is not more than forty-eight hours. The onset is usually sudden and characterized by fever, pain in the abdomen, and frequent stools, first containing mucus, and later consisting chiefly of mucus and blood. The movements increase in frequency and are associated with tenesmus, which becomes very marked. The movements may occur as frequently as every half-hour, and there is much straining. The tongue is coated with a white fur and there is excessive thirst; nausea and vomiting may occur. The abdomen is not distended, but there may be tenderness, especially over the colon. There are cramp-like pains in the abdomen. The spleen is not usually enlarged. The temperature rises to 103° or 104° F. It may run an irregular course and rise or fall before death. The pulse increases in rapidity (100 to 120 or even to 150) and becomes rapid and feeble. Urine is decreased and may contain albumin. Liver is not enlarged and *no liver abscess occurs;* moderate leukocytosis may be present. In very acute cases the patient becomes seriously ill within forty-eight hours, the movements increase in frequency, the pain is of great intensity, severe headache occurs, and the patient becomes delirious and dies on the third or fourth day. Lobar pneumonia, bronchopneumonia, acute bronchitis, and fibrinopurulent pleurisy may complicate.

In cases of moderate severity the symptoms abate, stools lessen in frequency, temperature falls, and within two or three weeks the patient is convalescent. In the subacute cases the attack may last many weeks or even months, and the patient have three to five bloody mucous stools in twenty-four hours, and become very emaciated. One of Strong's cases died on the sixty-fourth day. The Bacillus dysenteriæ is found in the stools and agglutinates with blood-serum.

**Other Clinical Types.**—The description just given applies to the types of bacillary dysentery such as seen in Japan, the Philippines, and the tropics, and the features of that in adults in temperate climates differ in no essential, except in many cases the symptoms are less severe.

Duval, as noted, has found one of the bacillary dysentery strains in sporadic cases in Philadelphia and elsewhere, and the probability is that most cases of non-amebic dysentery belong to this type.

The so-called acute catarrhal dysentery is unquestionably a sporadic form due to the Bacillus dysenteriæ. This is the more probable when we consider that in ileocolitis (dysentery) in infants we find a catarrhal type. In infants there are four types of lesion found on autopsy (Holt):

Follicular ulceration; catarrhal inflammation; catarrhal inflammation with superficial ulceration; membranous inflammation.

This last differs from the membranous type in adults, in that there is little pseudomembrane and no deep sloughing. Holt has well described these varieties.

Methods of serum diagnosis in the bacillary dysentery of infants are described by Lucas and Fitzgerald[1] at the laboratories of the Harvard Medical School. They advocate the conglutination reaction.

Diphtheritic dysentery is a type of the bacillary form with great necrosis and infiltration of the mucosa. It is believed that other types of bacteria are also often associated in the process. The pure dysentery bacillus, unlike the typhoid bacillus, does not lead to bacillemia or bacilluria (Herter, Bacterial Infections of the Digestive Tract). In the diphtheritic type, a mixed infection in which streptococci undoubtedly are prominent, the condition is unquestionably, in my opinion, a *general infection*, as shown by the complications.

The secondary diphtheritic dysentery is a common terminal event in many acute and chronic diseases. Vedder and Duval have demonstrated that the bacilli are present in these cases.

Peritonitis is rare, either through extension or by perforation. When it occurs about the cecal region, perityphlitis results, or when low down, periproctitis.

In 108 cases collected by Woodward, perforation occurred in 11.

Abscess of the liver is *very rare*. A few cases occurred in the Civil or South African War. In the tropics malaria and dysentery may coexist, and typhoid and dysentery coexisted quite frequently in the Civil War. In ordinary practice it is very rare.

**Complications.**—Acute bronchitis, pneumonia, bronchopneumonia, pleurisy, gangrene of the lung, albuminuria, meningitis, paralysis, paraplegia, in many cases due to a neuritis, are not uncommon; thrombosis of the cerebral sinuses, embolism (cerebral), rheumatic pains and swollen joints (analogous to gonorrheal arthritis), pericarditis, endocarditis, periproctitis, perityphlitis, and occasionally pyemic manifestations, such as pylephlebitis or abscess of the spleen, may occur. Anemic edema may be present in protracted cases. Chronic Bright's disease is an occasional sequel and intestinal stricture is *rare*. Persistent dyspepsia and irritability of the bowels may follow.

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[1] Jour. Amer. Med. Assoc., Feb. 5, 1910.

**Prognosis.**—In the very severe cases the prognosis is bad, the patient often dying within one to two weeks. This is especially true in epidemics. In milder cases convalescence begins by the end of the second week. Chronic cases may run weeks or even months, the patient becoming very emaciated. They may die at the end of several months. Convalescence is slow in any but the mild cases.

**Treatment.**—*Prophylaxis.*—The same precautions should be exercised as regards boiling drinking-water, avoiding green vegetables, cleanliness of the hands, destruction of flies, disinfection of stools and linen, as are carried out in amebic dysentery and in typhoid fever.

*Medication.*—The bowels should be at once thoroughly cleared with castor oil, 1 to 2 ounces (30.0–60.0), or with magnesium sulphate or sodium sulphate, 1 to 2 drams (4.0–8.0). Buchanan has had excellent results by the sodium sulphate treatment; he gives 1 dram (4.0) sodium sulphate four to even eight times a day for two to three days until blood and mucus have disappeared. He has treated 855 cases with 9 deaths—an excellent record.

The old ipecac treatment is still in considerable repute in tropical countries. No food is taken for three hours, then 20 drops of laudanum are administered and one-half hour later 20 to 60 grains (1.3–4.0) of pulverized ipecac. If this is vomited, the dose is repeated in a few hours. The salol-coated ipecac pill, as described under Amebic Dysentery, may be substituted.

In South Africa the saline treatment was often given combined later with the ipecac, and Washbourne reported good results.

Ringer recommends small doses of bichlorid, $\frac{1}{100}$ grain (0.00065), every two to three hours, and large doses of bismuth subnitrate have been used with success, at least ½ dram (2.0) and sometimes as much as 1 dram (4.0) every two hours daily for the first few days. The following I have found the most rational method of internal medication.

The bowels should first be thoroughly cleared by the administration of 1½ to 2 ounces (45.0–60.0) of castor oil in the adult, or by several doses of 1 to 2 drams (4.0–8.0) of magnesia sulphate or sodium sulphate; calomel, 5 grains (0.3), may precede this. A hot application should meanwhile be applied to the abdomen to relieve the pain, and if the latter be unendurable and there is considerable collapse, a very small hypodermic of morphin may be given, sufficient to render it endurable and not large enough to interfere with peristalsis. It is preferable not to administer opiates until thorough cleansing of the bowels has been accomplished. In fact, it is my belief that opium is given too frequently and in too large doses, so that material accumulates in the bowel which had better be expelled.

Bismuth subnitrate, which it is preferable, as a rule, not to use in the undermined ulcers of amebic dysentery, in this type is of great value. The dose should be large. After thorough clearing of the bowels, bismuth subnitrate alone or combined with equal parts of saccharated pepsin should be given.

At least 20 to 30 grains (1.3–2.0) of this mixture or even 20 grains (2.6) of bismuth subnitrate every two to three hours. Bismuth subcarbonate, 10 to 15 grains (0.6–1.0), can be substituted. Bismuth subgallate, 5 to 10 grains (0.3–1.6) t. i. d., or bismuth salicylate, 10 grains (0.6) t. i. d., can be substituted in place of some of the usual doses of the other preparations; thus, for example, bismuth subgallate and bismuth subnitrate could alternate. Bismuth subnitrate, 30 grains (2.0), mistura cretæ, 2 drams (8.0), is an excellent combination in one dose, given every three or four hours. The tannin preparations, such as were suggested in amebic dysentery, can be given, three doses substituted for three of the bismuth in the course of twenty-four hours. Thus, if seven doses of bismuth were given in twenty-four hours, give three of tannin and four of bismuth. Tannalbin, 5 to 7½ grains (0.3–0.5), or even 15 to 30 grains (1.0–2.0), can be given four or five times daily. It can be administered with grated chocolate or beaten up with an egg or with a little strained rice gruel. Tablets, 5 grains (0.3), can also be secured. If the pain is severe, small doses of opium may have to be given, with the precautions noted. Children should receive proportionately small doses of all remedies. The opiates are described under Diarrhea. Urotropin, I believe, might be of service; urotropin, 5 to 10 grains (0.3–0.6), combined with equal quantities of sodium benzoate, given four to six times daily. It might lessen the toxemia and be of service in the cases of mixed infection.

Tenesmus should be relieved first by thorough bowel irrigation, and after this, if it continue, by inflation with a small amount of $CO_2$ (Rose's method), or by a small injection of starch-water and laudanum, 15 minims (0.888), with tincture of belladonna, 10 minims (0.59). Opium should never be given first, thus tying up the foul secretions.

Strychnin, nitroglycerin, or camphorated oil by hypodermic may be required. Local treatment is of paramount importance. Zinc, sulphocarbolate of zinc, and silver nitrate are preferable in the chronic cases. Hypodermoclysis, or even infusion, may be required in shock or severe sepsis. Proctoclysis is of use in the latter.

If there is much tenesmus, a preliminary suppository of belladonna extract, $\frac{1}{3}$ grain (0.022), alone or, rarely, combined with $\frac{1}{8}$ grain (0.008) cocain, or the injection of $\frac{1}{8}$ grain (0.008) cocain in 1 ounce (30.0) water, may rarely be necessary. The best method is to insert a recurrent tube and gently irrigate with normal saline solution at 110° to 115° F., or cold at 40° to 50° F., and then, forcing the tube in still further, continue the antiseptic irrigation.

I have found the recurrent method of irrigation with a double tube (preferably) or two tubes (soft catheters) of great value in these cases, the patient's hips or foot of the bed being elevated. The patient is gradually accustomed to the fluid, and the quantity allowed to run in before permitting return can slowly be increased and there is not the endeavor to expel the solution. A single injection of any size can often not be given.

On the other hand, 1 to $1\frac{1}{2}$ liters (quarts) can be injected by the method of Musgrave in amebic dysentery in some cases.

In the *acute cases* acetozone, alphozone, hydrogen peroxid, and permanganate of potash are of especial value, in the same strength noted below as employed in amebic dysentery.

Delafield has secured excellent results by the use of bichlorid of mercury (1: 10.000), using 2 quarts (liters), but cautiously, with my recurrent tube. I have had good results by this method, but it should never be given by ordinary enema.

Acetozone (1 : 1000), hydrogen peroxid (1 : 10), alphozone (1 : 1000), and permanganate of potash (1: 1000) are excellent.

Mucol, 1 dram (4.0) to 1 quart (liter) or even stronger, has been employed. This preparation contains the chlorid, borate, bicarbonate, and benzoate of soda, with zinc sulphocarbolate and essential oils.

Several irrigations or enemata may be necessary daily, depending on the tenesmus or character of the stool: one or two antiseptic, and the rest of normal saline solution, or flaxseed or gum-arabic.

If mucus and blood increase, a cathartic is again indicated, and I believe it should be given every three days in any event.

*Carbolic acid should never be employed* for irrigation.

*Chronic Dysentery.*—Bismuth is of value, Nitrate of silver injections are of service—10 to 30 grains (0.6–2.0) to 1 pint (500 c.c.)—and inject 2 to 3 pints if possible; if there is irritation, follow it with an injection of normal saline solution.

Protargol or argyrol (1: 500) are useful, and *often preferable to* silver nitrate.

Normal saline or normal saline with oil of peppermint, 10 minims (0.59), can be used between the antiseptic injections, or an injection of flaxseed tea or gum-arabic solution. The latter are soothing.

**Diet.**—*Acute Cases.*—Fluid diet, broths, gruels, barley- and rice-water, beef juice, sanatogen, egg-albumen, and milk well diluted. Somatose is of use. Personally I prefer milk-free diet.

In the *chronic cases* fruits and green vegetables should be avoided and constipating food given if there is diarrhea. The weight of the patient should be increased by administration of fats. The *general diet rules* should be similar *to those in amebic dysentery.*

*Serum Therapy.*—Shiga immunized horses and produced a polyvalent serum from which he claims to have reduced the mortality of dysentery in Japan from 35 to 9 per cent.

Flexner has prepared a serum, but Holt states results in children have proved disappointing.

*Complications* should receive appropriate treatment.

**Treatment of Dysentery Bacilli Carriers.**—Mayer[1] states that attempts to dispossess these bacilli by internal medication have failed. He advises isolation until the germs disappear from the stools. This has been found to require from three to seven months. The author refers chiefly to cases in troops where sanitary measures are more available and where the danger of lighting up new epidemics is more marked. In addition to isolation, the stools require immediate disinfection. It has been found that if the period of isolation is too short, bacilli which have temporarily disappeared from the stools may reappear. Meanwhile treatment should be continued and the effort made to prevent the condition from becoming chronic. Urotropin and sodium benzoate (āā gr. x), four times daily, and high irrigations with acetozone (1 : 1000) may be continued.

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[1] Münch. medizin. Wochens., Dec. 6, 1910.

## CHAPTER XXVI

# TYPHOID FEVER—PARATYPHOID FEVER—BRILL'S DISEASE

A MERE outline of this disease will be given for the purpose of differential diagnosis. Typhoid may be defined as a general infection caused by the Bacillus typhosus, characterized anatomically by hyperplasia and ulceration of the intestinal lymph-follicles, swelling of the mesenteric glands and spleen, and by parenchymatous changes in the other organs, such as in the kidneys, liver, etc. Clinically the disease is marked by fever, rose-colored eruption, abdominal tenderness, tympanites, diarrhea, and splenic enlargement, but these symptoms are inconstant.

**History.**—Louis, in 1829, gave the name to the fever. Gerhard, in 1837, first clinically differentiated between typhoid and typhus.

**Etiology.**—General prevalence. It prevails in temperate climates. Imperfect sewage and contaminated water-supply favor the distribution of the bacilli; filth, overcrowding, and bad ventilation aid in lowering the resistance of the individual. *Fingers*, *food*, and *flies* spread the disease from the infected person. It is prevalent in England, Wales, India, parts of Germany, and in the United States, where it occupies about fourth place in the mortality list. It has been one of the great scourges of armies, more destructive than powder and shot. The mortality in the South African and Spanish-American wars was greater from typhoid than from bullet wounds; among the American troops, about one-fifth of the soldiers in the national encampments had this disease. The former inefficiency of our sanitary methods was a blot upon our government. In great contrast to this were the remarkable results secured by the Japanese in the recent Russo-Japanese War, where, in comparison with our own records, typhoid and dysentery were practically negligible factors.

*Season.*—The disease is more prevalent in the autumn, though frequent cases occur during August.

*Sex.*—Both sexes are equally liable, but males are more frequently admitted to the hospitals.

*Age.*—Typhoid is a disease of youth and early adult life, the greatest susceptibility being between the ages of fifteen and thirty. Cases are rare over sixty. It is not infrequent in children. Infants are rarely attacked.

*Immunity.*—Not all exposed take the disease. One attack usually protects; two attacks have been described, and occasionally three.

*Bacillus Typhosus.*—The researches of Eberth, Koch, and others have shown that the disease is due to a special micro-organism. It is a short, thick, flagellated, motile bacillus with rounded ends, in one of which, sometimes in both, there can be seen a glistening round body, probably an area of degenerate protoplasm. It grows readily on various nutritive media and must be differentiated from the Bacillus coli.[1] The organism fulfils all the requirements of Koch's law. Cultures are killed in ten minutes by a temperature of 60° C. The direct rays of the sun destroy them in from four to ten hours' exposure. Bouillon cultures are destroyed by carbolic acid (1 : 200) and by bichlorid (1 : 2500) solution. Toxins have been isolated from the bacilli.

*Distribution in the Body.*—The typhoid bacilli may be demonstrated in the circulating blood, and have been found in the urine, stools, sweat, sputum, and in the rose-spots. They occur in the mesenteric glands, spleen, and gall-bladder, and have been found in almost all the organs, even in the muscles, uterus, and lungs. Cultures from the intestines show that few, and frequently none, can be cultivated from the rectum up to the cecum. Above this they are numerous. They have been found in the mucous membrane of the stomach, also in the esophagus, and on the tongue and tonsils, and have been isolated from endocardial vegetations, meningeal and pleural exudates, and from foci of suppuration in various parts of the body. They may be present in the stools of healthy people who have lived in close association with typhoid patients.

*Bacilli Outside the Body.*—They retain their vitality for weeks in water. This is further demonstrated from infection by ice, in which they exist for several months, and live for some days in butter from infected cream. They may live in the upper layers of the soil for months; in street dust for a month or more; on linen for two months; and on wood for a month.

*Modes of Conveyance.*—Aërial transmission is not probable. Fingers, food, and flies are the chief means. House infection is difficult to avoid unless finger contamination is carefully eliminated. Such epidemics have been reported at the Johns Hopkins Hospital. Epidemics have occurred from infection of water, milk, and food, such as celery and uncooked vegetables which have grown in infected soil. Raw oysters are recorded as a cause of epidemics. Bedbugs and fleas may be carriers. Water infection is the most common cause. There is some evidence that the digestive tract is not the only portal of infection for typhoid fever, but that the bacilli may enter by way of the throat, notably by the tonsils (Herter).

**Morbid Anatomy.**—*Intestines.*—A *catarrhal condition exists throughout the large and small intestine.* Specific changes occur in the lymphoid elements, chiefly in the lower end of the ileum. Four stages are described:

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[1] Paratyphoid infection is referred to under Diagnosis.

1. Hyperplasia, involving Peyer's patches in the jejunum and ileum and, to a variable extent, those in the large intestine. They are swollen, grayish-white in color, and may project from 3 to 5 mm. The solitary glands may project to a variable extent.

There is hyperemia of the follicles; later an increase and accumulation of the cells of the lymph-tissue, which may infiltrate the adjacent mucosa and muscularis; the blood-vessels are compressed, which gives a white anemic appearance to the follicles.

This process reaches its height from the eighth to the tenth day, and then undergoes either resolution or necrosis.

2. Necrosis. When the hyperplasia is marked, resolution is no longer possible. The blood-vessels become choked; there is a condition of anemia; then necrosis occurs and sloughs form, which must be separated and thrown off. This process is always more intense toward the ileocecal valve. The necrosis is variable, it may pass deep into the muscular coat, and even perforate the peritoneum.

3. Ulceration. Sloughing is effected from the edges inward, and results in the formation of an ulcer, the extent of which is directly proportionate to the amount of necrosis. The muscularis usually forms the floor of the ulcer.

4. Healing. The mucosa extends from the edge and a new growth of epithelium is formed, as are the glandular elements. The healed ulcer is depressed. Healing is never associated with stricture.

*Large Intestine.*—The cecum and colon are affected in about one-third of the cases, and the solitary glands are sometimes enlarged.

*Perforation.*—About one-third of the deaths, Scott's statistics state, are due to perforation. It occurred in 3.6 per cent. of all cases.

The German statistics are much lower; in Munich only 5.7 per cent. of deaths are due to perforation. Among 1500 cases at the Johns Hopkins Hospital there were 43 of perforation; 20 were operated on, and 7 of these recovered. The site of the perforation is usually in the ileum, within 12 inches of the ileocecal valve. It may be from a pin-point to large size.

*Death from Hemorrhage.*—This occurred in 12 of 137 deaths in Osler's 1500 cases. He could not find the bleeding vessels.

*Mesenteric glands* are hyperemic and swollen. Necrosis is common; abscesses may occur, causing peritonitis or hemorrhages.

*Spleen* is enlarged, infarction is not infrequent. Rupture may occur.

*Bone-marrow.*—Same changes occur as in lymphoid tissue, and there may be foci of necrosis.

*Liver.*—Parenchymatous degeneration present. Liver abscess has been found, also acute yellow atrophy. Pylephlebitis may occur.

*Gall-bladder.*—Acute cholecystitis may be present.

*Kidneys.*—Cloudy swelling with granular degeneration is frequent; less commonly an acute nephritis; miliary abscesses or diphtheritic inflammation of the pelvis may occur; also infection by colon bacilli. With colon bacilli infection, chills, rise of temperature, and acute renal symptoms occur.

*Bladder.*—Cystitis or diphtheritic inflammation may complicate. Orchitis, acute mastitis, and parotiditis are occasionally met with.

*Respiratory Organs.*—Ulcer of the larynx; edema of the glottis; diphtheritic inflammation; bronchitis; pneumonia; hypostasis; pleurisy; gangrene; abscess of the lung; hemorrhagic infarction and empyema may complicate.

*Circulatory Changes.*—Endocarditis, pericarditis, and myocarditis, endarteritis, arthritis of a peripheral vessel with thrombus formation may occur. Venous thrombosis is more frequent, especially of the left femoral.

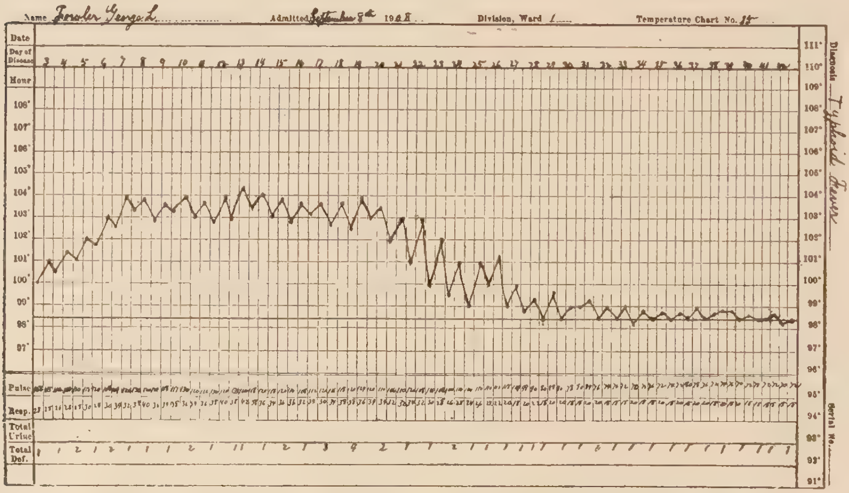

Fig. 246.—Typhoid chart.

*Nervous Symptoms.*—Meningitis is rare; optic neuritis may occur; the cause of aphasia seen in children is not positively known. Parenchymatous changes may occur in the peripheral nerves.

*Voluntary Muscles.*—The muscular substance, especially of the recti, pectorals, and adductors of the thigh, may undergo granular degeneration or hyaline transformation. Rupture, hemorrhage, or abscess have been found.

*Reflexes.*—The superficial skin reflexes of the abdominal walls are frequently absent or markedly diminished in typhoid fever. The return of these lost reflexes is usually synchronous with other evidences of recovery.

**Symptoms.**—Incubation, eight to fourteen days, occasionally to twenty-three days, during which period there are lassitude and inaptitude for work. Onset is rarely abrupt, with occasional chills.

There may be cough (bronchitis), epistaxis, headache, anorexia, diarrhea in many cases, but frequently early constipation, abdominal pain, and distention; and in some cases there is pain in the right iliac fossa. At the onset the patient usually takes to his bed.

During the first week there is in many cases a steady rise of temperature, the evening record rising a degree or more each day, and reaching 103° to 104° F. (Fig. 246), Variations of temperature are common. It may rise suddenly to 104° F. or may fall suddenly. It may be high A. M. and lower P. M.

The pulse is rapid, 100 to 110, full, but of low tension, and often dicrotic. The tongue is coated and white, the abdomen slightly distended and tender. Unless there is high temperature there is no delirium. The patient complains of headache and there may be mental confusion at night. The bowels are loose or may be constipated. At the end of the week the spleen becomes enlarged and the eruption appears in the form of rose-colored spots, seen first on the abdomen. They are raised, flattened papules, can be felt by the finger, and disappear on pressure. They come out in crops and may appear on the trunk or extremities. Desquamation may occur.

In the second week the symptoms become aggravated; the fever remains high and the morning remissions are slight, the pulse is rapid and loses its dicrotism. There are mental torpor and dulness; the lips are dry and the tongue may become dry. Tympanites, tenderness, and diarrhea, if present, become aggravated. The stools are described as "pea-soup." Death may occur at the end of this week from hemorrhage or perforation, but generally later. In the third week the pulse ranges from 110 to 130. The temperature shows marked morning remissions and there is a gradual decline in the fever. Loss of flesh is more marked and weakness is pronounced.

Diarrhea and meteorism may in some cases occur for the first time. Unfavorable symptoms are pulmonary complications, feebleness of the heart, delirium with muscular tremor, and acute tympanites. The tongue may become brown or brownish black and the lips and teeth be covered with sordes.

Special dangers are perforation and hemorrhage.

The fourth week convalescence begins. Temperature gradually becomes normal; diarrhea stops; tongue cleans; the desire for food returns. In severe cases the fourth and fifth weeks present an aggravated picture of the third, the patient grows weaker, pulse more rapid and feeble, tongue dry, and abdomen distended. He lies in profound stupor with low muttering delirium and subsultus tendinum, and passes the feces and urine involuntarily. Heart-failure and secondary complications are the chief dangers.

In the fifth and sixth weeks protracted cases show irregular fever, and convalescence may not set in until the fortieth day or longer. During this period, recrudescence of temperature may occur from errors in diet, constipation, or excitement; or a relapse (re-infection) may take place. With relapse there is a repetition of the ascent of the original fever. A rise in temperature from inflammatory complications usually has leukocytosis associated with it.

*Modes of Onset.*—As a rule, the *onset is insidious.* The following deviations may occur: Marked nervous symptoms, such as headache or cerebrospinal symptoms, with retraction of the head and convulsions; or mania; or stupor. Pulmonary symptoms, such as bronchitis, pneumonia, or pleurisy. Intense gastro-intestinal symptoms, with pain and vomiting, suggestive of poisoning, or in some cases simulating appendicitis. Acute nephritis may be the first symptom. Ambulatory form, in which the patient keeps about and attempts to work. This runs a severe course and often a fatal issue results. Chills may occur at the onset and may be followed by sweats, and they are present with complications. Variations from the typic temperature are common, the step-like ascent does not always occur, but the fever may rise suddenly. A sudden fall of temperature is suggestive of hemorrhage before blood appears in the stools. Peliomata, maculæ cerulæ—pale-blue or steel-gray spots—are sometimes present, due to lice. Erythema may occur. Bed-sores are not uncommon and boils are a troublesome sequel.

*Blood.*—*Leukopenia* (hypoleukocytosis) is present; lymphocytes are relatively increased. *Eosinophiles disappear* or are markedly diminished. The reappearance or increase of eosinophilia is a good prognostic sign. *Leukopenia* and *absence of eosinophiles* aid diagnosis. Hemoglobin and red corpuscles are reduced.

Severe meteorism is a danger-signal and predisposes to hemorrhage or perforation. Acute gastro-intestinal dilatation or acute ectasia alone may occur. Acute distention may even simulate perforation; and it is only possible to differentiate by relieving the distention by enteroclysis and lavage.

*Symptoms of Perforation.*—Sudden sharp pain, at times paroxysmal, often in the hypogastric region to the right of the median line; tenderness, sudden distention, and muscular rigidity. *This last is an important symptom.* There are shock, fall of temperature, pallor, sweating, and the Hippocratic facies. The temperature then rises, pulse rapid and feeble, respiration increases. Vomiting is often present. Leukocytosis and especially increase in the polynuclears occur. Percussion may show a flat note in the flank, due to exudate. Obliteration of liver flatness may be caused by tympany.

Abscess of the liver and cholecystitis may complicate. Gallstones in many cases are probably associated with the presence of typhoid bacilli in the gall-bladder. Under Lesions most of the complications are referred to. Loss of hair may take place.

Local neuritis, as in the arms, legs, or toes (tender toes), may occur. Multiple neuritis is a complication of canvalescence. Poliomyelitis, tetany, and hemiplegia have been reported. Typhoid psychoses may occur, also eye and ear complications, and retention of urine. Post typhoid anemia may be severe.

*Ehrlich's Diazo-reaction.*—This test is not absolutely diagnostic, as it occurs in miliary tuberculosis, in malaria, and occasionally in other acute disease, associated with high fever. It is of accessory value taken with other data. Bacilluria occurs in about one-third of the cases. Acute appendicitis may complicate.

*Osseous System.*—Periostitis, osteitis, caries, and necrosis are troublesome sequelæ of typhoid, as are arthritis and typhoid spine. Colitis, simple catarrhal or of a septic (diphtheritic) type, may complicate.

*Post typhoid septicemia and pyemia are not uncommon.* Furuncles, abscesses, and infarcts in various regions may occur. With children typhoid fever often runs a mild and irregular course.

**Diagnosis.**—The type of temperature, splenic enlargement, eruption, absence of leukocytosis (leukopenia), disappearance of the eosinophiles, together with Ehrlich's reaction, are the chief signs. Isolation of the typhoid bacilli from the blood, stools, and urine is diagnostic.

Widal's reaction is of positive value when found. It is generally not in evidence until the seventh to tenth day, sometimes not until convalescence, and occasionally not at all. Cabot claims over 90 per cent. reactions before the eighth day. Fortunately, it is present in about 90 per cent. of cases. Blood examination will differentiate between typhoid and malaria. Ulcerative endocarditis has been mistaken for typhoid, but the presence of the heart lesion and the streptococci in the blood are diagnostic. With acute miliary tuberculosis the temperature is irregular or intermittent. Tubercle bacilli may be found in the fluid by lumbar puncture, and there is an absence of typhoid bacilli in the blood-culture. The tuberculin test (ocular) or by injection is of value. Respiration is more rapid and there is more cyanosis. Widal is negative, but leukocytosis is common. With typhoid we have the Widal reaction; there is leukopenia, splenic enlargement is present, and the typic eruption. Intestinal grip may for a few days simulate typhoid, as may intestinal toxemia. The subsequent course, absence of Widal reaction, etc., clear the diagnosis.

The paratyphoid bacilli A and B may produce symptoms like a mild typhoid fever, and paratyphoid B may be found in meat-poisoning, and have a causal relation to the typhoidal symptoms. The Bacillus enteritidis (Gärtner) may also be found in meat-poisoning, and this bacillus, and also the Bacillus fæcalis alcaligenes, Herter shows, may incite symptoms simulating mild typhoid fever. Recent studies, however, have shown that these bacilli differ from typhoid bacilli in their agglutination reactions. Hemorrhage is rare and never alarming. Widal is negative. Moreover, the serum of patients ill with meat-poisoning regularly agglutinate known cultures of the causative organism, as does that of paratyphoid A. With Brill's disease cultures from the blood, urine, and feces are negative. Widal is negative. There is no intestinal hemorrhage. Leukopenia is absent.

**Prognosis.**—Mortality. Death-rate is variable, depending on the severity of the epidemic and when treatment has begun. Of recent years it has been from 5 to 20 per cent. Fat people stand typhoid badly. Meteorism, hemorrhage, high fever, and nervous symptoms give a bad prognosis.

**Prophylaxis.**—Care in *drainage* and *water-supply* are most important. These are the chief precautions to be taken in army camps. Vaccine therapy as a means of prophylaxis the writer also believes of great value in war. Undoubtedly this proved efficacious during the Boer War in the English army, being introduced by Wright. Russell[1] reports favorably on this method as a prophylactic in the U. S. Army, and refers to the work of Leischmann in the English army, and to the results secured in the German-African campaign. Russell advocates three preventive injections at ten-day intervals—the first injection of 500,000,000 dead bacteria, and the subsequent doses of 1,000,000,000 each. Raw milk, raw oysters, and uncooked fruit and vegetables should be avoided during an epidemic. Raw oysters in New York should generally be avoided, unless the source of the supply is known. The urine, stools, and sputum should be disinfected. The sputum can be collected in cloths and burned, and bichlorid (1 : 1000) or carbolic (1 : 20) used to disinfect the other discharges. About twice the volume of the disinfectant should be mixed with the stools, and it should be left to stand for two hours before emptying. For disinfection of the bath-water, ½ pound of chlorid of lime will render an ordinary bath of 200 liters sterile in one-half hour (Babucke). The nurse should wear a rubber apron and should wash it frequently with carbolic or bichlorid. When giving baths, rubber gloves, or especially *rubber finger-tips*, should be worn, and the hands thoroughly disinfected later. The hands have been found to be the source of transfer of the disease in about 90 per cent. of the cases,[2] which proves the necessity of isolating these cases. All bedding and the patient's night-dress should be soaked two hours in 1 : 20 carbolic and then boiled. A special cup, dish, spoon, etc., should be used for the patient, and disinfected on each occasion after using. It is best to keep them in the room and transfer the food from another vessel to the special cup or dish; if in a ward, after each feeding they should be placed in carbolic (1 : 20).

After recovery, the room should be disinfected. Osler shows that 1.81 per cent. of cases of typhoid at the Johns Hopkins have been of hospital origin. I have noted great carelessness in our hospitals as regards fly protection, seldom observing screening of the patient during the fly season. Typhoid cases should preferably be kept in a *special ward to avoid the danger of infection of other patients through carelessness*. Mabon demonstrated this at the Manhattan State Hospital.

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[1] N. Y. State Jour. of Med., Dec., 1910.

[2] Jour. Royal Army Med. Corps, Jan., 1907.

The method so often followed in our hospitals of treating typhoid in the general medical ward the writer believes most pernicious.

**Chronic Typhoid Distribution.**—Many cases are discharged from the hospitals while there are still typhoid bacilli in the urine and stools. Examinations of these discharges should be made.

Urotropin, 5 to 10 grains (0.3–0.6), with an equal amount of sodium benzoate t. i. d., is excellent to disinfect the urine. It may be necessary to increase it to four or five doses daily.

An interesting case of typhoid carrier of nearly seven years' duration, with five small epidemics to her credit, is reported by George A. Soper.[1] Rosenberger[2] gives a report on the literature of typhoid carriers.

**Treatment.**—*General Management.*—The patient should be in a light, well-ventilated room, confined to bed. This should be single, with a comfortable mattress, covered with a blanket, and a rubber cloth placed under the sheet. Nursing and diet are the essentials.

*Diet.*—There is a tendency among many practitioners to endeavor to increase the resisting power to typhoid by increased feeding, and who hold that for this purpose the patient should lose as little weight as possible. In some cases solid food has been administered; in others liquids, with considerable dextrose and cream.

W. Coleman[3] advocates milk, cream to a pint daily, bread and butter, milk-sugar, and eggs, giving from 4000 to 5500 calories per day, and reports good results. The author disagrees with a method which places upon diseased organs twice the labor which they perform in health, with the dangers incident to excessive feeding.

F. Meara[4] employs milk, cream, etc., in his diet, toasted bread, gruels, ice-cream, etc. He is more conservative as to forcing the quantities, but gives as much as the patient takes well, and diminishes the quantity if gastro-intestinal disturbances appear.

C. W. Strong[5] gives a *milk-free diet*, boiled rice, 6 to 7 ounces at a time, being the basis of his feeding, together with eggs, gelatin, bread, butter, broths, with milk-sugar and a little cane-sugar, up to 3500 calories a day. He secures best results by *omitting* milk.

*It has been thoroughly demonstrated* that no matter what the intake, the nitrogenous output in this disease is always markedly in excess.[6]

Typhoid is an *acute general infection*, and in such conditions all the digestive functions are abnormal. There is change in the character of the bile. Stölmkow[1] noted disturbances in the pancreatic juice; and I recently referred to the diminution[2] of motor power and the lessening or absence of hydrochloric acid during the high temperature of typhoid fever. The parenchymatous changes in the liver and kidneys interfere with elimination by these organs, and the associated intestinal catarrh causes further interference with the digestive functions. Excessive feeding as now advocated by some—3500 to 5500 calories—the writer holds prolongs the temperature, as much detritus will be found in the bowels, and after the emptying of the same, followed by proper milk-free diet, the temperature runs a lower course. Stuffing the patient is done at the expense of diminishing water, which the writer advocates in large amounts to eliminate the toxins. Seibert, in 1889, noted that the temperature and tympanites were lessened when milk was withheld, the fever falling to 99° or 100° F. on the ninth to twelfth days. Rectal irrigations he found of great value. He[3] gives rectal injections with 3 pints of warm normal saline solution, preferably several times a day, depending on the case, thus irrigating the bowel. During the first day of treatment cold water only is given as food. From the second day on, $\frac{1}{2}$ pint of strained rice, oatmeal or barley soup, containing the extract of $\frac{1}{2}$ pound of meat and the yelk of a fresh egg, well spiced, are given every three hours, five times daily. From the fourth day on, strained pea, lentil, potato, and tomato soup with rice, were added to the menu. Two or three zwiebacks were given with the soup at the end of the first week. Orange-juice was given in water three times daily. Egg-albumen was not given on account of the probability of forming toxins. Before each meal 15 to 25 drops of hydrochloric acid were administered in $\frac{1}{2}$ ounce of water. No alcohol was given except to topers, and camphorated oil was employed by hypodermic if stimulation was required. Cold baths were never employed, even in hyperpyrexia. Sponging was added if necessary. Opium was only used in bowel hemorrhage.

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[1] Jour. Amer. Med. Assoc., June 15, 1907.

[2] N. Y. Med. Jour., March 26, 1910.

[3] Jour. Am. Med. Assoc., Oct. 9, 1909.

[4] Amer. Jour. Med. Sci., Jan., 1911.

[5] Amer. Med., Oct., 1910.

[6] Finkler and Lichtenfeld, Central. für die Allgemeine Gesundheitspflege, 1902.

Lesser[4] has treated all fevers above 102.5° F. with water alone, and below this point with broths, rice-, and barley-water, also employing enteroclysis. Good results were secured by these methods in typhoid fever during the Spanish-American War.

The author's method is as follows: As gelatin lessens nitrogen excretion, and as it aids in preserving weight, and furthermore causes no putrefaction in the intestines, it is of some value as a food. The ingestion of 7.5 per cent. of the total heat requirement of the organism in the *form of gelatin spares 23 per cent. of the body's protein.* Thus, in a total of 2800 calories required by a man of 154 pounds (Chittenden), 210 calories in gelatin are necessary; 1 gram of gelatin contains 4.1 calories, so about 50 grams of gelatin are required, or 1½ ounces (48.0). The gelatin and cereal gruels approximate 2000 to 2500 calories, all that can be digested properly. Gelatin also lessens the tendency to hemorrhage.

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[1] Pflüger, Archiv. Physiologie.

[2] Med. Rec., June 20, 1908, and Amer. Med., May, 1909.

[3] Med. Rec., June 20, 1908.

[4] Ibid., Oct. 19, 1905.

Approximately, 1½ ounces (48.0) of gelatin in 12 ounces (375 c.c.) of water, gives a 12 per cent. solution. This gelatin solution can be slightly flavored with lemon, vanilla, or with sugar, and be given in divided doses. Strained rice, barley, oatmeal, and chicken broths, the sum total not over 1½ to 2 quarts (liters), are also to be given in divided doses every three hours, the last feeding no later than 9 P. M. This makes in all about 2 to 2½ quarts (liters) of nourishment. The yelks of raw eggs, 3 to 4 daily, are beaten in with the broths, and gruels and butter is added. Of this a trifle under ½ ounce represents 100 calories. Milk-sugar represents about 120 calories to the ounce, and it may be added to each feeding with broth if it seems to agree. Feeding should begin about 6 A. M., and the last nourishment at 9 P. M. (five feedings in all). Occasionally, one may give an additional dose of gelatin between the other feedings. Strained pea or strained potato soup may be given once daily in place of one of the gruels.

One should administer about 2500 calories daily if there are no digestive disturbances, and as low as 2000 in some cases. One should individualize in each case. The gelatin is of great value for the reasons stated. The various foods are alternated for variety. The *patient is not urged to eat.* Whenever the temperature reaches 103° F. or more, nothing but water is administered until it falls to that point. The *juice of several oranges* is given during the day. When the temperature falls to 99.5° F., the gruels are thickened considerably and the yelks of raw eggs increased to 5 or 6 daily.

At least 1½ to 2½ quarts (liters) of water, to which dilute sulphuric acid, 20 minims (1.184 c.c.), is added, are to be drunk by the patient during the twenty-four hours. Dilute nitromuriatic or dilute hydrochloric acid may be substituted for the dilute sulphuric acid.

For *the advocates* of milk, I would state that the *sour milks*, such as matzoon, bacillac, fermillac, koumiss, kefir, and lactone-buttermilk, are preferable to plain milk. Effervescence should be allowed to a great extent to pass off from koumiss before administering, and it is preferable to dilute it some with lime-water or water. Matzoon and the thicker sour milks should be diluted one-half with plain water, or Vichy that has become flat, to avoid distention. Milk, if administered, should be diluted one-half, preferably with barley-water or rice gruel. In 100 c.c. of milk is contained only 64 calories, or 640 calories per liter; 4 quarts (liters) of undiluted milk would not give more than 2800 calories required for a man weighing 154 pounds. The fallacy of pure milk-diet is thus demonstrated. Rice, barley, and oatmeal average about 350 *calories per* 100. If milk is given, its calorie value and digestibility are increased by these cereals. About 1½ quarts (liters) of milk, or sour milks thus diluted, could be given in twenty-four hours. I do not advocate their use. Carbonated waters while effervescing add to distention. Sanatogen, as much as 1 ounce (32.0) in divided doses, is of value, given in the broths.

*Bowels.*—The bowels are freely opened by calomel, 5 grains (0.3), or castor oil, 1½ ounces (45.0), on the first day, and thereafter hot saline enemata, 1½ liters (1500 c.c.), at 110° to 115° F., or if gas, enteroclysis (recurrent), 1 gallon is given A. M. and P. M. as routine. Hemorrhage, perforation, or appendicitis are the only contraindications. There is one exception to this rule: gentle bowel irrigation with a tube and funnel, with hot normal saline solution at 120° F. during hemorrhage, lessens tympanites and helps contract the vessels. Performed *by the physician*, if guarded by a hypodermic of morphin, ¼ grain (0.016), to prevent subsequent peristalsis, I believe, with Kaufmann, the procedure to be of value.

*Temperature.*—*Sponging.*—With diet and irrigation of the bowels *tub-baths* are rarely necessary. Cold sponging with alcohol and water, combined with friction when the temperature reaches 102.5° F. or over, generally suffices.

Strong[1] has secured reduction of temperature and relief of nervous symptoms by continuous irrigation of the bowel with cold normal salt solution with the Kemp tube, in one case with ice-cold water for an hour or more, where baths failed.

*Baths.*—I am not opposed to the Brand method as a scientific procedure for its additional effects on the pulse and on elimination. If the friction bath is given, it should be started ot about 90° F., never given below 70° F. Often the warm bath is preferable.

*Improvised Tub.*—One can improvise a bath-tub, *in fact, build it about the patient*, by rolling up heavy blankets, in the shape of bolsters, forming the shape of a bath-tub. A rubber horse-blanket or heavy rubber sheet is then slipped under the patient and the edges brought over the rolled blankets. Water at the desired temperature is poured gently over the patient, friction being employed at the same time. The water is ultimately mopped up with large sponges, and the patient dried in a blanket. The head can be flexed and lie on a pillow just beyond the edge of the tub, the shoulders lying within it. The feet can also extend beyond the improvised tub, being wrapped in blankets. This method allows one to use a short improvised tub in the patient's bed and avoids practically all manipulation and lifting.

The Nauheim bath (Triton salts), advocated by William H. Thomson, especially if friction is combined, is superior to the Brand bath. In Fig. 247 is a portable tub, weight 5 pounds, excellent for private work.

Strychnin, $\frac{1}{60}$ to $\frac{1}{30}$ grain (0.00108–0.0021), or Hoffmann's anodyne, 1 dram (4.0), may be required in the bath. The average duration of the bath should be twenty minutes, longer if no reduction of high temperature occurs. If the patient looks blue or shivers, he should be removed.

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[1] Amer. Med., Oct., 1910.

*Medicinal Treatment.*—Antipyretics should be avoided. Though Chantemesse has reported some results from serum-therapy, it is doubtful whether anything of value has yet been obtained, though interesting data have been reported on the injection of bacterial vaccines by Walters and Eaton.[1]

Anders[2] believes vaccine therapy should receive a more extended trial, and advocates it as a prophylactic, during convalescence, to prevent relapses, to combat local infections from the typhoid bacillus, and for the removal of typhoid bacilli from the feces and urine. He questions its safety in the severe types.

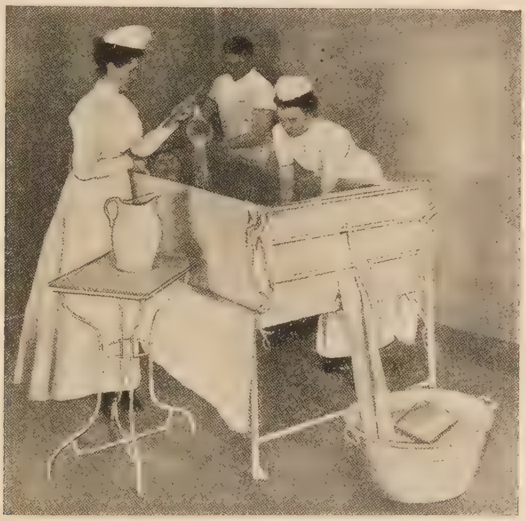

Fig. 247.—Chambers' portable bath-tub.

Bismuth subnitrate or subcarbonate, in a dose of 15 to 20 grains (1.0–1.3), combined with the same quantity of saccharated pepsin, I employ every three hours, on an average of four doses a day. The bismuth helps control the ulcers. As urotropin causes disappearance of the bacilli from the urine, it would seem a logical remedy. Urotropin, 5 to 10 grains (0.3–0.6), given preferably with equal quantities of sodium benzoate in Vichy which has become flat, is the best method. It should be administered three or four times a day or oftener, from 30 to 60 grains daily. I have apparently seen some benefit in lessening the temperature and tympanites by this method. Acetozone (1:1000), given in divided doses, about 1 to 1½ quarts (liters) per day, has been favorably reported in some cases; each dose is flavored with orange juice.

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[1] Med. Rec., Jan. 16, 1909.

[2] Jour. Amer. Med. Assoc., Dec. 10, 1910.

*Proctoclysis.*—Riesman[1] suggests the value of proctoclysis in the severe cases, as it increases urinary flow and lessens nervous symptoms. As an aid to elimination of the toxins the writer believes it may prove of value. It is also useful when perforation and sepsis occur.

*Tympanites.*—Hot fomentations and turpentine stupes are employed. The ice-bag is best in many cases. A rectal tube may be inserted, or a soapsuds enema containing oil of turpentine, 1 dram (4.0), be given. Spirits of turpentine, 10 to 15 minims (0.592–1.184), can be given three or four times a day, or resin turpentine, 3 grains (0.194), four times a day, or oil of cinnamon, 3 to 5 minims (0.178–0.296), every two hours. Charcoal, 5 grains (0.3), bismuth subnitrate, 15 grains (1.0), beta-naphthol, 3 grains (0.194), or ichthalbin, ichthoform, or formidin, 5 grains (0.3), every three to four hours, may be substituted. Acute distention is relieved by enteroclysis. As acute dilatation of the stomach is often associated, lavage is also of value. This is especially true in distention with active hemorrhage.

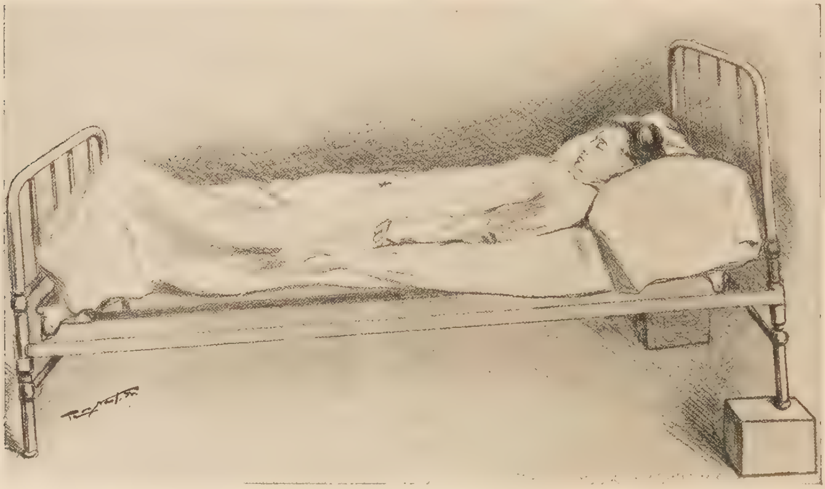

Fig. 248.—Postural treatment for acute dilatation of stomach and intestines in typhoid fever.

A thorough bowel action should at once be secured if there is no hemorrhage. Eserin, $\frac{1}{60}$ grain (0.00108) by hypodermic, may be of value for this purpose. Several doses may be required at two-hour intervals, two or three in all, each guarded with strychnin, $\frac{1}{60}$ grain.

As I order magnesium sulphate or citrate, 1 dram (4.0) every second or third day, in addition to the rectal irrigation, tympanites is rare.

In Fig. 248 is illustrated the correct position to relieve pressure from tympanites; pulse and respiration lessened 20 points as a result, and the tympanitic area in the thorax diminished 4 inches.

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[1] Jour. Amer. Med. Assoc., Jan. 29, 1910.

*Diarrhea.*—The bismuth preparations, chalk, and occasionally a little opium (see chapter on Diarrhea), may be required, and the enemata should be stopped for twenty-four hours, if the movements are excessive.

*Constipation* does not occur with the methods described.

*Hemorrhage.*—Morphin, $\frac{1}{4}$ grain (0.016), by hypodermic, is indicated, and the ice-bag should be immediately applied. Then lactate of calcium, 15 grains (1.0), is given with 2 to 4 drams (60.0–125.0) of 5 to 10 per cent. gelatin solution; chlorid of calcium, 10 grains (0.6), may be substituted. Thereafter, lactate of calcium, 10 grains (0.6), with 2 ounces (60.0) of 10 per cent. gelatin every four hours. Ernutin, 5 minims (0.296), may be given by hypodermic.

William H. Thomson recommends the following:

| | | |
|---|---|---|
| ℞. | Pulv. opii<br>Argenti nitratis | āā gr. v (0.3); |
| | Resin turpentine | ʒij (8.0); |
| | Liquor potassii | ʒj (4.0); |
| | Licorice pulv. | q. s. —M. |

Divide into 60 pills.

Sig.—Two pills every four hours. They may be given for a few doses at two-hour intervals.

Large doses of opium should be avoided, as they obscure symptoms.

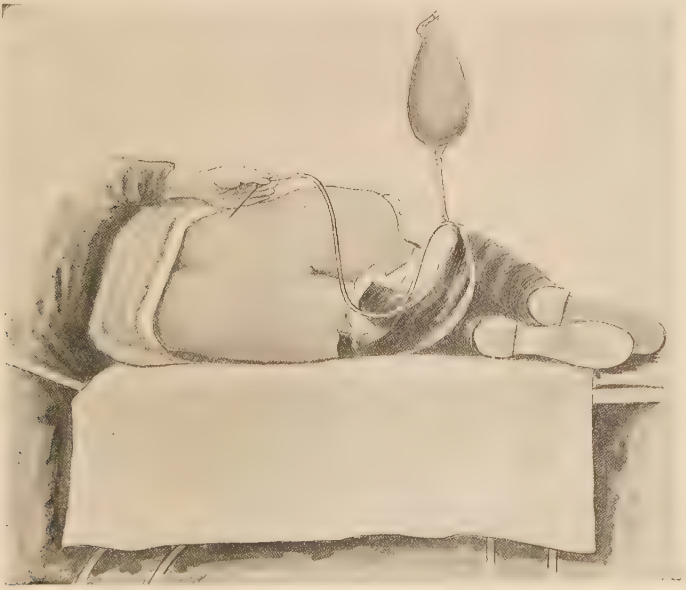

Fig. 249.—Hypodermoclysis in the iliolumbar region.

Adrenalin (1 : 1000), 5 to 10 minims (0.296–1.592), has been advocated by hypodermic, but it may increase pulse-tension too markedly. The administration of human serum may be indicated in severe hemorrhage.

Hypodermoclysis, preferably in the iliolumbar region, as in Fig. 249, with normal salt solution, may be required, or even infusion.

At any time, on the appearance of blood in the stool or of suspected hemorrhage, stop enteroclysis and baths (if they are being given) *at once.*

*Perforation and Peritonitis.*—Early operation is indicated.

*Heart Stimulants.*—Strychnin sulphate, $\frac{1}{60}$ to $\frac{1}{30}$ grain (0.00108–0.0026), every three or four hours by hypodermic, or,

℞. Pulv. camphor.......................... gr. viiss (0.5);
Sterile almond oil......................... ♏xx (1.184).—M.

Sig.—One dose every four to six hours by hypodermic may be required, especially if there are cardiac complications.

Aromatic spirits of ammonia or Hoffmann's anodyne, in 1-dram (4.0) doses, are useful in emergency.

Caffein citrate, 1 to 2 grains (0.065–0.021) every three or four hours, is of service; it may be given by hypodermic in the form of sodii salicylate of caffein or sodium benzoate of caffein.

Some advise alcohol, 8 to 12 ounces (250–375 c.c.), in divided doses, but, like Seibert, I have not found it necessary. Large doses of tincture of digitalis or digitalone, 5 to 15 drops every four hours, may be added in the case of alcoholics. Careful stimulation with drugs, I believe, causes less strain on the kidneys..

The former views as to the value of alcohol as a food, or its power to increase the capacity for work, seem, by scientific research, to be pretty thoroughly exploded. Its use in the arctic regions, where food of high calorie value is at a premium, has been found to be deleterious. In sudden emergency, as a heart stimulant, it is of value, but its prolonged use as a circulatory tonic has been shown to be harmful. Proper individual dosage is difficult to estimate, and the strain on the already damaged eliminating organs in typhoid fever is severe.

Professor Frederick S. Lee,[1] in a series of experiments, finds that in small quantity ethyl alcohol does not appear to exert any action on frog's muscle; while in medium quantity it increases the rapidity of contraction and relaxation, and increases the working time, *i. e.*, delays fatigue. In large quantities it exerts an unfavorable action, the reverse of that caused by medium quantities of the drug, *i. e.*, it *hastens fatigue.* The advocates of the use of alcohol in typhoid fever administer it in considerable quantities. Moreover, the author wishes to call to the reader's attention that alcohol interferes with the oxidation process.

*For Nervous Symptoms.*—Warm or cool packs with ice-bag to the head, and at times bromids or opiates, are indicated.

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[1] The Action of Alcohol on Muscle, Amer. Jour. of Physiol., 1902, viii, p. 61.

*For Headache.*—Cold applications and the bromids are indicated.

*For Sleeplessness.*—Sulphonal, trional, or veronal, 10 grains (0.6).

*Bacilluria.*—Urotropin and benzoate of soda, of each 10 grains (0.6), every three hours by mouth, or by enema if nausea, should be given in colon bacillus infection.

Care should be taken to guard against bed-sores. Tender toes should be protected from the weight of the sheets, and hyperextension of the feet should be avoided. In some cases a water- or air-bed or an old sheepskin spread under the patient, as suggested by Thomson, may be required.

*For Renal Insufficiency.*—Cream of tartar lemonade: Cream of tartar, 1 dram (4.0); juice of 2 lemons; saccharin, 1 grain (0.063); water, 1 quart (liter). Drink in divided doses during the day. Recurrent enteroclysis with normal saline solution at 120° F. and proctoclysis are useful. Caffein citrate, 5 grains (0.5), t. i. d., is of value, or an equivalent of a soluble preparation of caffein by hypodermic. Hypodermoclysis may be necessary, or proctoclysis.

Colitis should receive treatment as described in that chapter. Delafield[1] has secured results in membranous colitis by irrigation with 2 quarts (liters) of bichlorid of mercury (1 : 10,000) with the recurrent tube.

Complications should receive appropriate treatment.

*Convalescence.*—It is usually preferable to have a normal temperature for a week before commencing with solid food. Soft-boiled eggs, milk-toast, jellies, and a little scraped beef should be first tried.

In some *prolonged cases*, with temperatures of 99° to 100° F., cautious feeding may be attempted with the above materials. I have seen the temperature fall as a result.

Bacilluria I believe a frequent cause of slight persistent temperature. This should receive treatment as already described. The patient should first sit up for a brief period about the tenth day of normal temperature.

During convalescence the urine should be particularly examined for colon and typhoid bacilli, and urotropin, 10 grains, with an equal quantity of sodium benzoate, should be given three or four times daily. The urine should *be free from typhoid bacilli* before discharging the patient.

## PARATYPHOID FEVER

This is a term applied to a group of diseases which, in their clinical course, may somewhat resemble typhoid fever.

**Etiology.**—Paratyphoid fever is caused by the micro-organisms known as Bacillus paratyphosus A and Bacillus paratyphosus B, which differ in their agglutination reactions, that is, the blood-serum of the patient agglutinates the cultures of the causative organism. The organisms are isolated from the blood, urine, or feces. They are intermediate between the Bacillus typhosus and the Bacillus coli. In the epidemics abroad, Bacillus paratyphoid B is recovered from much more frequently than the paratyphoid A, which seems more common in America.[1] Recently, however, in an epidemic in Virginia, an epidemic from paratyphoid B, in all 35 cases, has been reported by Hoskins.[2] The Bacillus paratyphosus B also has an etiologic relationship to bacterial food (meat) poisoning, and the question has arisen whether there is a common infectious disease, assuming an acute form as meat-poisoning and a subacute form as paratyphoid fever. The Germans, in fact, assert that meat, particularly beef, is the habitat of the germ of Bacillus paratyphoid B, and that the fever is produced by toxins generated by these bacilli in ingested beef. They describe one variety resembling ptomain-poisoning, occurring with prodromal symptoms, such as bronchitis, nose-bleed, headache, malaise, and abdominal tenderness. There is at first a high temperature, which subsides after the administration of calomel. Subsequently, the temperature curve is very little above normal, though tympanites, gurgling in the right iliac fossa, rose-spots, enlarged spleen, abdominal soreness, and increased pulse-rate are present. This variety lasts seven to eleven days.

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[1] Enteroclysis, Hypodermoclysis, and Infusion (Kemp).

A second variety is characterized by a higher temperature and symptoms simulating typhoid fever, and lasts eleven to fourteen days, and in several cases three weeks. Proescher[3] and Roddy have conducted some interesting investigations on the paratyphoid A and B bacilli. These types of paratyphoid bacilli can readily be differentiated from the Bacillus typhosus and the Bacillus coli.

*Mode of Infection.*—Ingestion of meat containing the germ of the Bacillus paratyphoid B is claimed as a cause of this type, while the usual methods of conveyance are the same as of typhoid fever, such as the food, water, by the fomites, urine, fingers, and flies.

**Pathology.**—There are splenic enlargement and intestinal ulcerations, resembling those of dysentery rather than of typhoid fever. There is involvement of the solitary and agminated follicles and of the mesenteric glands.

*Incubation.*—The incubation is generally shorter than that of typhoid, though in Hoskins' cases it averaged in 50 per cent. nine to eleven days. The prodromal symptoms are languor, headache, torpor, and, in some cases, nose-bleed and muscular pains. There may be discomfort in the abdomen and constipation, though occasionally a diarrhea. The headache is often quite severe, and the prodromata are usually shorter and more sudden than with typhoid. There is bronchitis in some cases, coming on shortly, the headache is constant, and some chilliness is complained of in some cases, particularly in the extremities. The temperature frequently rises more rapidly than in typhoid, at times there being a high initial temperature, even to 104° F. The morning remissions *are more marked than in typhoid fever.* Paul[1] reports, in fact, a series of cases in which there was a daily remission nearly to normal. The pulse is rapid, 120 or more, but there is usually no dicrotism. Delirium rarely occurs. Nausea is frequently present, but vomiting is not very frequent. Tympanites is frequently present, though not as marked as in typhoid. There may be abdominal pains in the lower abdomen, though some cases are reported of severe gastric pain. Constipation is the rule, though diarrhea sometimes occurs. In Hoskins' cases the stools were at first normal, later, green tinted with yellow, and later, watery, with the peculiar tint noted. The urine is scanty and high-colored, with, at times, albumin and casts. Intestinal hemorrhage may sometimes occur, but it is not as severe as with typhoid, and is usually not recurrent. Perforation has not been reported. Bronchitis, as noted, is quite frequent. The tongue becomes thickly coated, particularly toward the center, and the edges may remain red and granular. The tongue is moist and does not dry and crack as with typhoid, and there are *no sordes.* The spleen is enlarged, but generally not as markedly as with typhoid. In many cases there are severe pains in the muscles and nerves. The skin is at first dry, but later becomes moist. Rose-spots appear on the third or fourth day, and may be seen in successive crops over the abdomen, chest, and back. In Hoskins' cases there was dilatation of the pupils which persisted throughout the disease. In his cases also day dreaming, illusions with no delirium, were reported in 25 per cent., and most of his patients were children.

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[1] H. Fox, Univ. Penna. Med. Bull., 1905, vol. xviii; and Proescher and Roddy, Jour. Amer. Med. Assoc., Feb. 6, 1909.

[2] Jour. Amer. Med. Assoc., March 19, 1910.

[3] Archiv. of Int. Med., March, 1910.

Some of the cases of paratyphoid infection, as already noted, are more of a ptomain type, a high initial rise of temperature with nausea and vomiting, with a rapid subsidence of temperature after the administration of calomel.

**Course.**—The course of paratyphoid is more frequently rather short, the highest temperature being reached on the third or fourth day, remaining high for three or four days, and then abating by lysis. It may occasionally be prolonged, though usually it is much shorter than typhoid, and relapses are rather rare.

**Complications.**—Bronchitis often accompanies it. Neuritis, nephritis, and cardiac disease are not as liable as with enteric fever. Intestinal hemorrhage is quite rare and never alarming. Perforation has never occurred. Parotiditis, arthritis, otitis, and osteomyelitis have been reported.

**Diagnosis.**—The onset of paratyphoid fever is more abrupt, the initial headache is more severe, the temperature rises more rapidly, the daily remissions are more marked, the tongue remains moist, there are no sordes, and intestinal hemorrhage is not frequent and is not apt to recur. Perforation has never been reported. The Widal reaction is absent. The specific agglutination reactions occur to Bacillus paratyphoid A or B, and the causative micro-organisms can be cultivated from the feces, urine, blood, and from the rose-spots. These all serve to differentiate from typhoid fever.

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[1] N. Y. Med. Jour., Oct. 22, 1910.

With Brill's disease the cultures from the blood, urine, and feces are negative. Widal reaction is negative. The type of fever is peculiar. There is no intestinal hemorrhage and rapid convalescence occurs.

**Prognosis.**—Most patients recover, though a fatal result may occur.

**Treatment.**—This is essentially the *same as typhoid fever*. The same precautions should be taken regarding safe-guarding the urine, feces, etc. Urotropin, 10 grains (0.6), with benzoate soda, 10 grains (0.6), should be given four or five times a day to disinfect the blood. Autogenous vaccines may be employed of the specific bacilli found in each case.

## BRILL'S DISEASE

**Definition.**—This is an acute infectious disease of unknown origin and pathology, characterized by a short incubation period, a period of continuous fever, intense headache, apathy, and prostration, a profuse and extensive maculopapular eruption, the fever ceasing at the end of between one and two weeks, either by crisis or lysis.

**History.**—The attention of the medical profession was first called to this disease by Nathan E. Brill,[1] who demonstrated that it possessed peculiar characteristics which differentiated it from typhoid and paratyphoid fever. He has made a study of 255 cases.[2] Ziegel[3] reports 3 cases.

**Incubation.**—This varies from a sudden onset to fourteen days.

**Etiology.**—The disease is infectious, the cause as yet undiscovered. There is no positive evidence as yet that the disease is communicable, as in Brill's 255 cases members of the same family or occupants of the same house were never attacked. W. Coleman, however, has observed 4 cases from the same family and household. Clinically the disease bears more resemblance to a mild typhus than it does to typhoid fever, and in a mild epidemic of typhus it would be difficult to differentiate. Its non-epidemic character, its usual non-communicability, its uniform picture, and the usual absence of grave and fatal cases are against the view that it is a modified typhus. Brill,[4] however, reports a fatal case.

Males are more commonly affected than females, and it is more frequent between twenty and forty years. The largest number of cases occur in summer.

**Symptoms.**—After a few days, marked by malaise, loss of appetite,

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[1] N. Y. Med. Jour., Jan. 8 and 15, 1898; Amer. Jour. Med. Sci., April, 1910.

[2] Amer. Jour. Med. Sci., Aug. 1911.

[3] Med. Rec., June 25, 1910.

[4] Amer. Jour. Med. Sci., Aug. 1911.

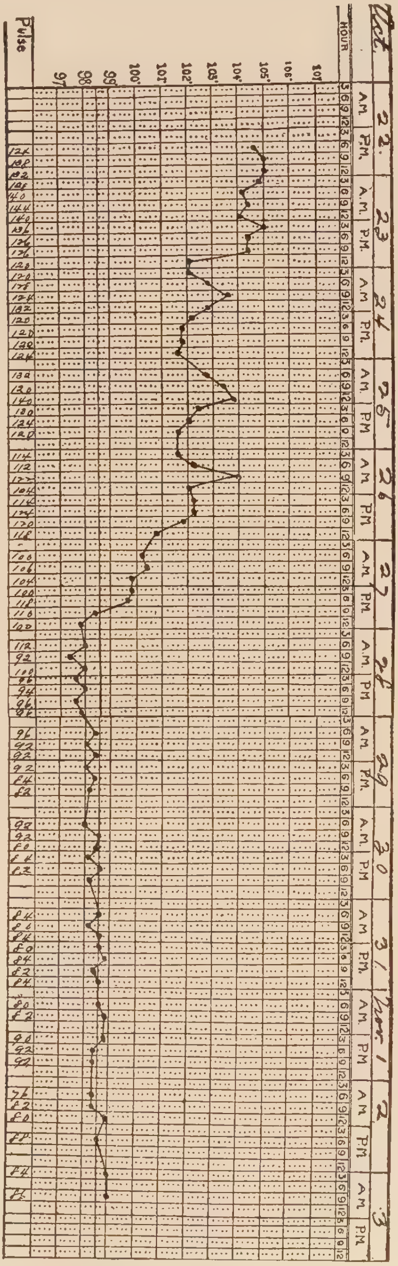

Fig. 250.—Temperature curve in Bril's disease (Ziegel's case).

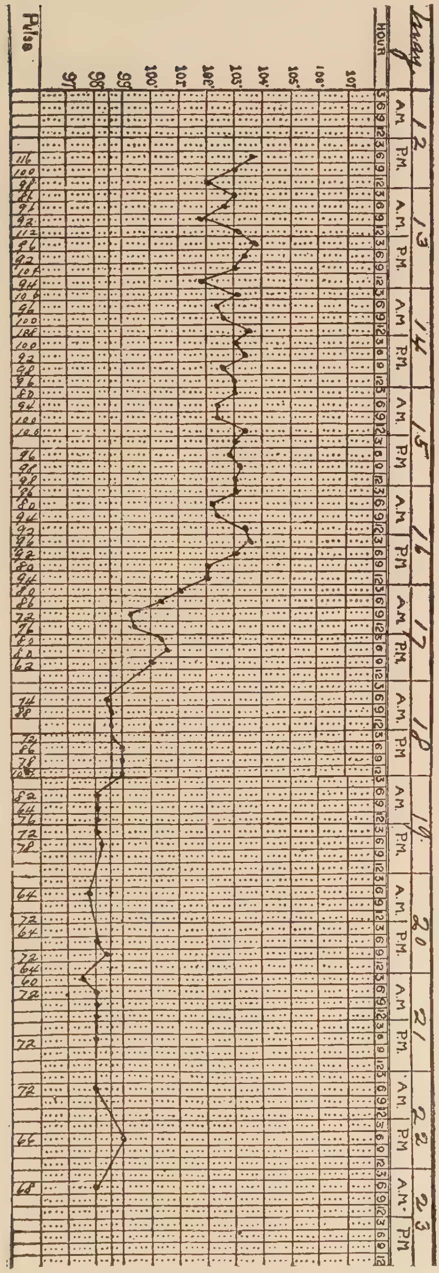

Fig. 251.—Temperature curve in Bril's disease (Ziegel's case).

nausea, and slight headache, the invasion is usually abrupt, ushered in with a chill or chilly sensations, followed by severe headache and a high fever. Occasionally there is vomiting, usually general pains, or pains in the back. The headache becomes intense and there is marked prostration, with a rising temperature. The temperature curve reaches its height in two to three days, and, with but slight daily remissions, remains constant. The temperature is shown in Figs. 250 and 251. The eyes are dull and suffused and the face flushed. The patient is dull and prefers to be let alone. The tongue is coated and moist. On the fifth or sixth day an eruption appears on the abdomen and back, and spreads rapidly to the chest and to the extremities. It is sometimes seen on the neck, palms, and soles in addition. The rash is profuse but discrete, and sometimes small patches are formed. It is maculopapular in character, dull red in color, slightly raised, and the spots usually have an oval indistinct outline. They fade somewhat under pressure, but do not disappear entirely. At times hemorrhagic spots are interspersed with the other lesions.

The eruption fades rapidly at the time of defervescence, but its remains are seen for some days as brownish stains.

The bowels are obstinately constipated. The spleen is frequently enlarged. Herpes labialis is sometimes present. Leukopenia is exceptional; at times there are 9000 to 11,000 leukocytes, though often the absolute and differential leukocyte count is normal unless there are complications. About the twelfth to fourteenth day the fever and other symptoms disappear and rapid convalescence follows.

**Complications.**—Bronchitis develops early in some cases. Bronchopneumonia is rare, in which event marked leukocytosis occurs. Meningismus, rigidity of the neck, contracted pupils, and bilateral Kernig's sign are sometimes present. Phlebitis has been observed once and otitis media once.

**Diagnosis.**—The absence of the Widal reaction and negative blood, stool, and urine cultures exclude typhoid, paratyphoid, and typhoid colon, or intermediate infections. Leukopenia, which occurs in typhoid, is absent in Brill's disease. The character of the onset, absence of intestinal hemorrhage, type of fever, character of the eruption, and rapid convalescence exclude typhoid fever. Its apparent noncommunicability, the uniformity of its type, and absence of fatal cases seem to exclude typhus fever.

The occasional meningismus which occurs suggests meningitis, but lumbar puncture shows its absence.

Influenza can be excluded by the history and by the speedy recovery from Brill's disease.

**Prognosis.**—So far one death has occurred.

**Treatment**—As a matter of precaution, the stools, urine, bedding, etc., are disinfected, and the same prophylactic measures taken as with typhoid fever. Sponging or friction baths should be employed to relieve temperature. Plenty of water should be given and the diet should be fluid, preferably strained broths and gruels. One may employ sour milks, such as matzoon with Vichy, koumiss, lactone-buttermilk, bacillac, etc., or equal parts of milk and lime-water.

The bowels should be opened freely with calomel, 5 grains (0.3), and a daily movement should be secured. For headache, cold applications and the bromids are indicated.

For the muscular pains the following is of value:

| | | |
|---|---|---|
| ℞. | Phenacetin | gr. iij; |
| | Acetanilid | gr. ij; |
| | Caffein citrate | gr. $\frac{1}{4}$.—M. |

Sig.—One capsule three or four times a day.

Brill[1] finally concludes that, though inoculation experiments with animals were a failure, clinically the disease resembles a mild typhus, and that it may eventually prove to be an attenuated modification of this disease. The writer describes Brill's Disease under the typhoidal group, for the purpose of differential diagnosis.

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[1] Amer. Jour. Med. Sci., Aug., 1911.

# CHAPTER XXVII

# INTESTINAL HEMORRHAGE; INTESTINAL ULCERS; DISEASES OF THE BLOOD-VESSELS (EMBOLISM AND THROMBOSIS)

## INTESTINAL HEMORRHAGE

It would seem advisable to refer to the various causes of intestinal hemorrhage for the purpose of diagnosis. Among such are dysentery, typhoid, yellow fever, malarial poisoning, ulceration from various causes, cancer, scurvy, purpura, traumatism, volvulus, intussusception after reduction of a strangulated hernia, excessive use of laxatives, hemorrhoids, injury (traumatism), venous hyperemia of the intestines due to diseases of heart or lungs, stasis or obstruction of the portal system, such as cirrhosis of the liver, injury from dried scybalæ, ankylostoma, isolated venous varicosities, arterial aneurysms of the intestinal wall, aneurysms in adjacent arteries, as of the hepatic, from which blood may enter the bile-passages and be passed by the bowel. One case is reported resulting from punctures in the intestines made by a male round worm.

A peculiar type of enterorrhagia occurs with no anatomic changes, but there is probably an alteration in the walls of the blood-vessels. Thus, intestinal hemorrhages have occurred in phthisis when there were no ulcers.

Intestinal hemorrhages in pernicious anemia, leukemia, scurvy, morbus maculosus, septicemia, icterus, phosphorus-poisoning, intermittent fever, and erysipelas probably belong to this class. Amyloid degeneration may be complicated by intestinal hemorrhages. Some believe vicarious hemorrhage may take the place of menstruation.

There may be collapse, with all the symptoms of internal hemorrhage, and blood may or may not pass from the rectum. The blood may be bright red, brown, or like coffee-grounds.

No visible blood may appear in the stools, and it may be detected only by tests for occult blood.

Microscopic examination may show the presence of red blood-corpuscles and hematin.

**Treatment.**—The immediate hemorrhage should be treated as described under Hemorrhage in Typhoid, and the cause should be determined and treated.

## ULCERS OF THE INTESTINES

### DUODENAL ULCERS

In considering the subject of duodenal ulcers, it is necessary to briefly classify the various types which occur. They are as follows:

1. Simple duodenal ulcer, or peptic ulcer of the duodenum, or chronic duodenal ulcer (Moynihan).
2. Duodenal ulcer due to burns or scalds.
3. Duodenal ulcer due to the uremic condition.
4. Tuberculous ulcer of the duodenum.
5. Duodenal ulcer due to Melæna neonatorum and to purpura.
6. Embolic or thrombotic ulcer of the duodenum, as with appendicitis.
7. Amyloid ulcer of the duodenum.
8. Duodenal ulcers in infectious diseases, as in pneumonia (rare), typhoid, erysipelas, and varioloid.
9. Duodenal ulcers have been reported in isolated cases of acute pemphigus and pellagra.
10. Syphilitic gummatous ulcer at the pyloric ring, involving the stomach or duodenum, or both.

### SIMPLE DUODENAL ULCER

(*Synonyms.*—Duodenal Ulcer; Ulcus Duodeni Pepticum—Leube; Round or Perforating Ulcer; Chronic Duodenal Ulcer—Moynihan.)

The earliest mention of duodenal ulcer occurs in the London Medico-Chirurgical Transactions of 1817, vol. viii, p. 232.

The condition is characterized by a defect of the mucous membrane of the duodenum, usually tending to run a protracted course, though in some instances it may be acute. Mayo[1] believes, however, that acute ulcers are usually toxic. One may say, in general, that duodenal ulcer presents the appearance and characteristics of ulcer of the stomach.

**Etiology.**—The cause is generally believed to be the action of the acid gastric juice upon the mucous membrane of the duodenum, whose nutrition and vitality have previously been impaired as a result of circulatory derangement. Chlorosis seems to play no part in its production.

Codman[2] advances the theory that more or less obstruction is produced in some cases by the pressure of the superior mesenteric artery on the duodenum, and as a result the secretions of the pancreas and liver may at times be thrown backward on the *first part of the duodenum*, which is unfitted to withstand the long-continued action of these secretions, since the mucosa is *histologically and developmentally* different from the rest of the duodenum, and is more closely allied to that of the stomach.

The author believes that if pressure obstruction from the superior mesenteric artery were a factor, one would frequently find duodenal ulcer associated with splanchnoptosis, which, according to some, favors chronic mesenteric traction. The writer has seen no preponderance of duodenal ulcers in such cases.

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[1] Boston Med. and Surg. Jour., April 6, 1911.

[2] Ibid., Nov. 25, 1909.

Mayo believes the acid thyme attacking the mucosa of the duodenum to be an important factor, and that when the chyme has been neutralized by the alkaline juices of the duodenum it ceases to produce irritation. He shows that, owing to anatomic formation, the alkaline juices reach a higher level in females than in males, and hence, the lesser number of ulcers of the duodenum in females. R. T. Morris[1] believes that toxic endarteritis, or toxic spasm of the terminal arteries in the duodenum, to be an explanation of the cause of duodenal ulcer in most cases. Cobweb adhesions, interfering with motility and leading to acid hypersecretion with local injury of the mucosa, he deems factors in other cases, or that colon bacilli in the terminal blood-vessels may produce an infarct.

**Frequency.**—There are wide discrepancies in the opinions of various observers as to the frequency of duodenal ulcer. The writer is now referring to the peptic type. Trier places it as 1 to 9 to the gastric as to frequency; while Andral gives 1 duodenal to 40 gastric ulcers; and Starke, 1 duodenal to 12 gastric. Moynihan believes duodenal ulcers to be quite common; and Wm. J. Mayo has demonstrated that many pyloric ulcers are found to have their origin in the duodenum, and in many cases it is impossible to differentiate between the two conditions. Codman believes duodenal ulcer to be more frequent than gastric ulcer, and to be even as common as acute appendicitis. Deaver[2] holds that duodenal ulcer outnumbers gastric ulcer in the proportion of 2 to 1, and probably more. The writer believes that undoubtedly many cases have been diagnosed as *gastric ulcer*, which were *duodenal ulcers* lying close to the pyloric ring. Ulcer of the duodenum is more frequent than is generally supposed, but not as frequent as gastric ulcer, in the author's opinion.

Moynihan holds that chronic duodenal ulcer, as far as concerns the cases coming to the surgeon, is a more frequent disorder than is ulcer of the stomach.

**Age.**—It occurs most frequently between the ages of fifteen and sixty. It occurs in the various decennial periods as follows in Moynihan's[3] cases:

| Years. | |
|---|---|
| 1 to 10 | None |
| 10 to 20 | 3 |
| 20 to 30 | 37 |
| 30 to 40 | 56 |
| 40 to 50 | 45 |
| 50 to 60 | 27 |
| 60 to 70 | 11 |
| Age not stated | 7 |

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[1] Amer. Jour. of Surg., April, 1911.
[2] N. Y. Med. Jour., March 18, 1911.
[3] Duodenal Ulcer, W. B. Saunders Co., 1912.

This table refers to the peptic type of duodenal ulcer. With melena neonatorum duodenal ulcer is not so uncommon in infants.

The youngest was seventeen years of age and the oldest sixty-seven. These were the ages given at the time of operation and not at the commencement of the symptoms. Many of the patients who were over forty had had symptoms for some years. Case 163, age forty-nine, had had symptoms for forty years, or they dated from the age of nine (see page 412, Moynihan[1]). The operative findings in this case appear to the writer as an ulcerative process originating in the *gall-bladder* and *not in the duodenum.*

Some cases of duodenal ulcer have been reported by other writers in *young children from one to ten years of age*, and also a number of cases among the newborn. In the latter, melena neonatorum or thrombosis of the umbilical vein, with a deposit of the thrombus in a duodenal vessel, with a resulting infarct, are the factors. Hereditary syphilis, with local ulcer of the duodenum, has been reported. Küttner[2] reports death in a child four years of age from duodenal ulcers. The postmortem showed duodenal ulcers, pseudomembranous colitis, and parenchymatous nephritis. These are not the true peptic duodenal ulcer and should not be thus classified.

Gastric ulcers occur in young children, but less frequently than the duodenal ulcers described.

The average age of death in 127 fatal cases was thirty-eight years (Rolleston), but the Fenwicks show that in the acute cases, 68 per cent. proved fatal between fifteen and thirty years; and in chronic cases between thirty and thirty-five years.

**Sex.**—Mayo reports 61 per cent. of duodenal ulcers as occurring in males. In an analysis of 186 cases, Moynihan reports its occurrence in 137 males, or 73.6 per cent.; females 49, or 26.4 per cent.; Weir, 30 women in 176 cases; Collins, 52 women in 257 cases. The ratio is variously given as 5 men to 1 woman, to 3 men to 1 woman. *The proportion of males affected by duodenal ulcer is considerably greater than that of females.*

**Variety of Ulcer.**—Among Moynihan's 137 male patients, there were 107 cases in which duodenal ulcer occurred alone, and 30 cases in which there were both gastric and duodenal ulcers. Of the 49 female patients, 32 had duodenal ulcer, and 17 had both duodenal and gastric ulcers.

**Pathology.**—William Mayo classifies duodenal ulcers surgically as indurated and non-indurated. In the former case they have a callous margin. The base is formed usually of the intestinal muscle, with scar tissue. There may be adhesions to adjacent organs, particularly to the omentum. Moynihan finds the majority free from adhesions. He believes that the peptic duodenal ulcer is usually chronic. Others classify duodenal ulcer as acute and chronic.

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[1] Duodenal Ulcer, 2d ed., W. B. Saunders Co., 1912.

[2] Berlin. klin. Wochensch., Nov. 9, 1908.

Melchior[1] discusses chronic duodenal ulcers and reports an acute case of duodenal ulcer, the hemorrhage occurring two days after operation for sarcoma of the thigh. He quotes 9 similar cases, and believes there is a diathesis which goes with a reduction in the vital resistance of the wall of the duodenum to the peptic action of the gastric juice. The writer, however, thinks the occurrence following operation rather suggestive of an embolic ulcer of the duodenum and not of the true peptic ulcer.

**Characteristics of the Ulcer.**—The duodenal ulcer resembles the gastric ulcer in its clean, punched-out appearance. In its earliest stage the ulcer is circular; later it may be oval or oblong. The crater of the ulcer is deep in proportion to its width (Fig. 252).

In some cases there seems to be steps leading down the side of the ulcer from the lumen of the gut to the base of the ulcer. This ridging or "terracing" is seen quite frequently. The walls of the ulcer are usually thick and indurated. The outer surface (peritoneal) is white and appears like a cicatrix as a rule. At times it may be red, vascular, and mottled with blood-stained spots. In the older ulcers the base is pearly white and puckered in the center, which is depressed and hard. They feel hard and dense to the fingers, and look smaller from the mucous than from the serous aspect. In others the entire breadth of the anterior wall of the duodenum may be occupied by a dense, fibrous mass, which extends even to the posterior surface. It may even have a keloid appearance. The ulcer may be star shaped, the center being drawn in. In some, "pouching of the gut" is produced, a piece of the duodenal wall being almost separated from the rest, so that a diverticulum is formed.

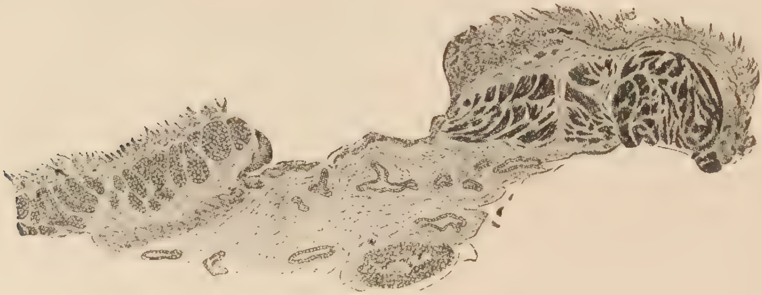

Fig. 252.—Duodenal ulcer (Le Tulle). On the right, the pyloric sphincter; on the left, the duodenum, with its mucous membrane studded with inflammatory nodules, the glands of Brünner, the submucosa, and the muscular coat uniformly invaded by the inflammatory lesions; in the center, the ulcer cut out perpendicularly, invading the muscular coat and resting on the thickened peritoneum, cicatricial tissue with large open vessels and lymphatic glands chronically inflamed (Gaultier).

In the majority a healthy margin of the bowel lies between the

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[1] Berl. klin. Wochens., Dec. 19, 1910.

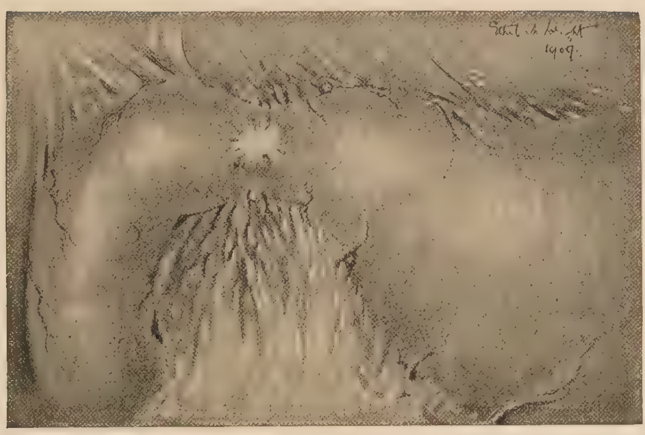

Fig. 253.—Duodenal ulcer. The usual position and size of the ulcer are well shown (Moynihan).

ulcer and the pylorus, but the lesion may extend to, within, or through the pylorus.

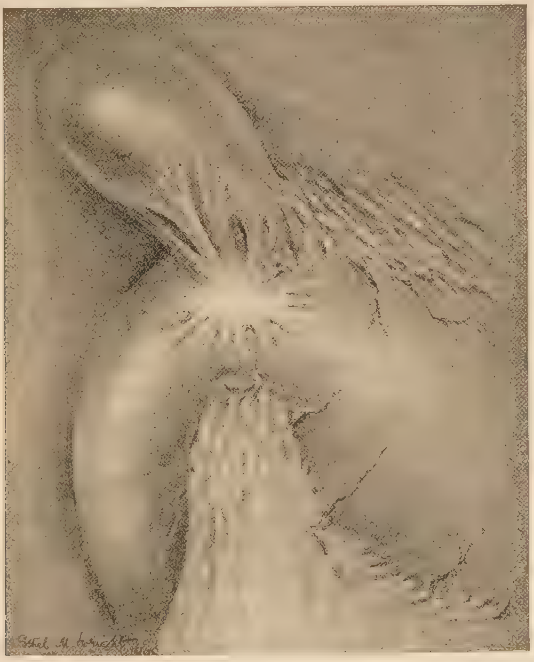

Fig. 254.—Duodenal ulcer. Showing adhesions to the gall-bladder. The radiating scar is not infrequently seen (Moynihan).

Mayo has shown that the gastric margin of the ulcer may be the starting-point of a carcinomatous growth. The occurrence of a malignant change in a simple duodenal ulcer is extremely rare, while it is quite frequent in gastric ulcer. In Fig. 253 is shown the

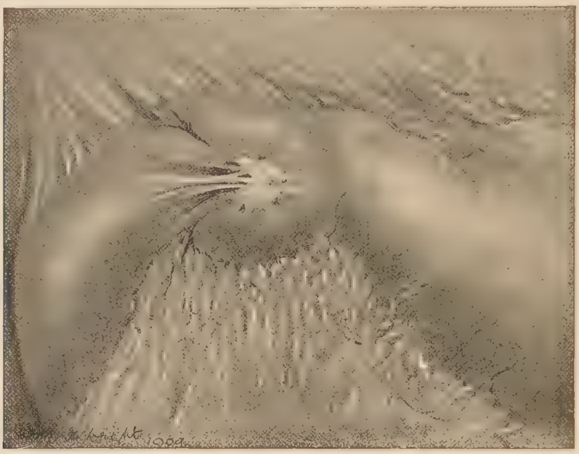

Fig. 255.—Duodenal ulcer. Showing the puckering up of the scar, which leads to "pouching" (drawn during operation upon the case) (Moynihan).

usual position and size of a duodenal ulcer. In Fig. 254 we have an ulcer with adhesions, and in Figs. 255 and 256 a "pouching ulcer."

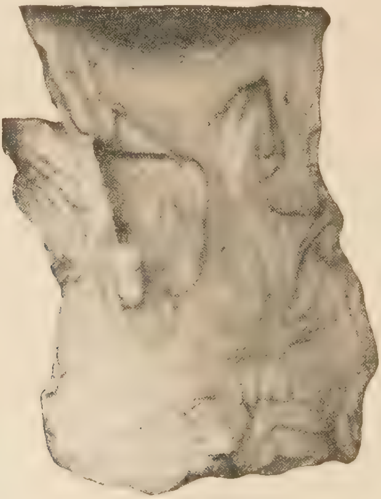

Fig. 256.—Chronic ulceration of duodenum with formation of a pouch (Moynihan).

**Site of the Ulcer.**—In at least 95 per cent. of all cases the ulcer lies within the first portion of the duodenum; that is, within 1½ inches of the pylorus. In Collins' cases, 262 in all, the ulcer was found in the first portion of the duodenum in 242 cases; in the second part, in 14; in the third, in 3; and in the fourth, in 3. In the cases in which the ulcer is "tucked back," adherent to the liver or posterior wall of the abdomen, in which event the pain comes on three or four hours after food instead of two hours, though the ulcer may apparently be situated in the second part of the duodenum, if the position of the pyloric vein be noted, it will be seen that the ulcer is within ½ to ¾ inch from the pylorus. This vein (Fig. 257) is an important landmark, and generally runs a little to the gastric side of the pylorus, and its recognition enables the operator to at once determine where the stomach ends and the duodenum begins. The vein runs upward from the greater curvature, is thick and short, and may often be met by a smaller vein descending from the lesser curvature. Usually there is no visible anastomosis. The ulcer is *generally situated on the anterior wall* of the duodenum, midway between the upper and lower border, about ½ inch beyond the pylorus. This part of the duodenum is apparently prone to attack, possibly because the chyme directly impinges there as it is expelled through the pylorus. Occasionally the ulcer is found on the upper or lower border and rarely on the posterior surface. One case of circular ulcer dividing the duodenum and opening into a local abscess is recorded by Meunier[1] (Fig. 258).

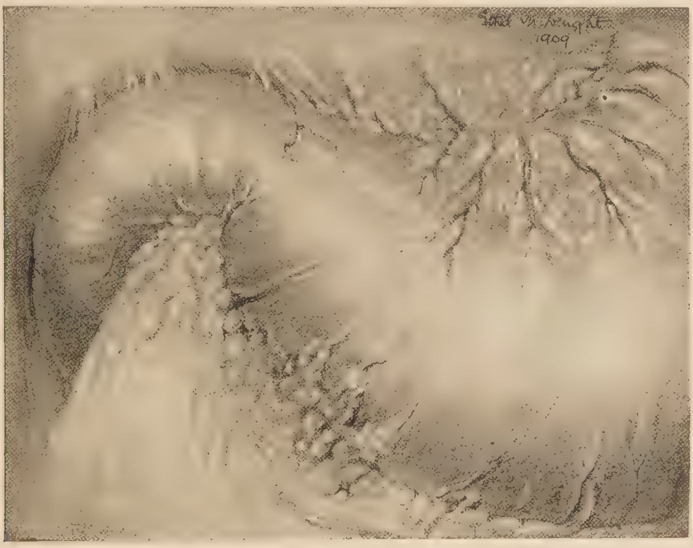

Fig. 257.—The vein which shows the position of the pylorus ("pyloric vein") (Moynihan).

**Recurrences.**—The recurrence of attacks in duodenal ulcer may be due to the healing and breaking down of a single ulcer or to the development of new ulcers. The former is more frequent, as in only 10 to 20 per cent. of cases is more than one ulcer found. Occasionally a large ulcer may be due to the merging of one small ulcer into another and so on. A large mass of fibrous tissue may occasionally be found. There is usually one ulcer, from the size of a lentil to a dollar.

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[1] Bull. Soc. Anat., 1893, i, 488.

**Number of Ulcers.**—In about 10 to 20 per cent. of cases, as noted above, the ulcers are multiple. Occasionally, from two to four or more ulcer scars are seen on the anterior surface of the duodenum, together with a new active ulcer. Old ulcers and new ones are found side by side; and when two ulcers are present, they are generally almost touching. One may lie on the posterior wall, opposite the anterior ulcer. When the gut is empty, they seem to be in apposition; and Moynihan suggests the term of "kissing" or contact ulcers (Fig. 259). When several ulcers are present they are usually found in the first part of the duodenum. As many as nine ulcer scars have been seen within the space of 1½ inches.

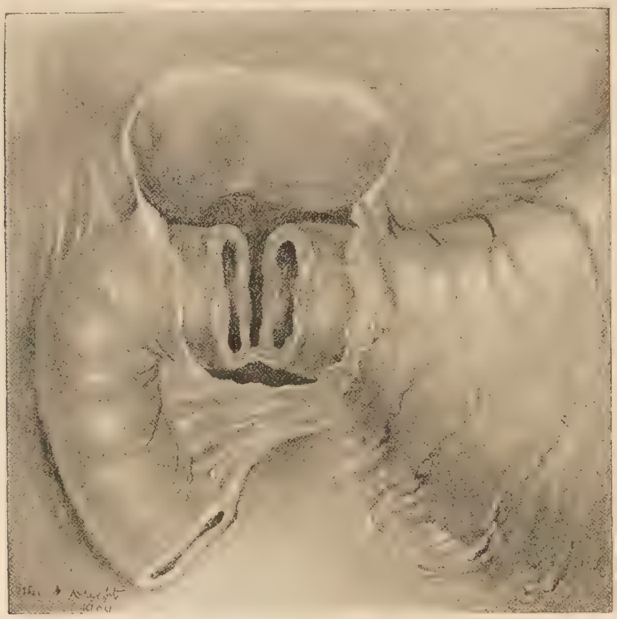

Fig. 258.—Circular ulcer of duodenum. Perforation into a localized abscess (Moynihan, after Meunier).

**Complications.**—Stenosis of the duodenum near the pylorus, or even at a greater distance, may occur, with resulting dilatation of the stomach and the symptoms of benign stricture, the same as in the stenosis of gastric ulcer. The stricture of the duodenum may be thin and narrow, the bowel appearing as if a string had been tied about it ("hour-glass"), or the stricture may be long, tortuous, and markedly indurated. In Figs. 260 and 261 we have illustrated the hour-glass condition.

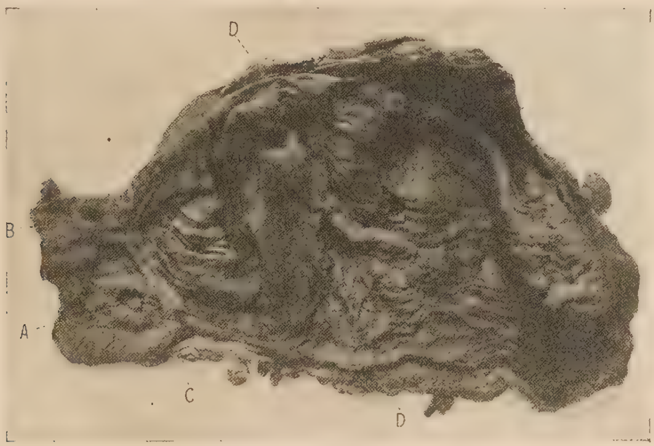

Fig. 259.—A, Perforating ulcer on anterior surface of duodenum; B, "kissing ulcer" on posterior surface; C, pyloric ring; D, cut edge of lesser curvature of stomach. Note the position of the ulcers immediately outside the pylorus.

The specimen was removed from a woman aged thirty, who died about two hours after admission to the Royal Victoria Hospital, Belfast, May, 1909. No operation was undertaken. From a photograph kindly given to Mr. B. G. A. Moynihan by Dr. A. B. Mitchell, Belfast (Moynihan).

Jaundice, with marked inanition and distention of the gall-bladder and inflammatory conditions therein, may result from stenosis from ulcer which blocks the common bile-duct, so that pancreatic carcinoma is suspected. Atrophy of the pancreas or chronic

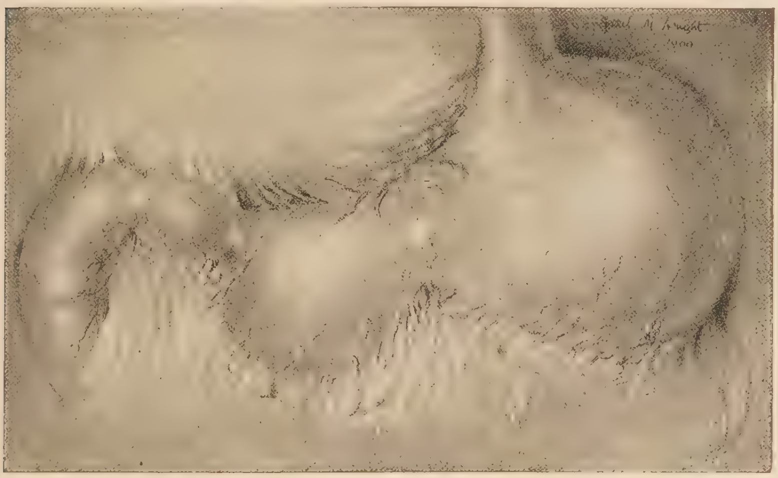

Fig. 260.—Hour-glass stomach and duodenum (Moynihan).

pancreatitis may take place from closure of the pancreatic duct. The type of hemorrhage is usually severe with the pancreatitis. Fatal hemorrhage may occasionally occur.

In acute cases quite frequently perforation and general peritonitis result, with death from shock or peritonitis. If the process is slower, there may be a circumscribed peritonitis with abscess. There may be adhesions to the omentum and with other organs and ulcerations involving the liver, gall-bladder, pancreas, aorta, portal vein, or hepatic artery. Subphrenic abscess may result. Thrombosis of the portal vein has also resulted from the deep cicatrization of an ulcer. The blood-vessels of the duodenum have frequently been eroded, chiefly the gastroduodenal, the pancreaticoduodenal, and the right gastro-epiploic arteries. In one case the hepatic artery was eroded, and in a few cases there was an aneurysmal dilatation of the vessel at the point of rupture. Perforations of the aorta, portal vein, and superior mesenteric vein have been reported. Hepatic abscess may result from duodenal ulcer. Two cases of duodenocolic fistulæ are recorded.

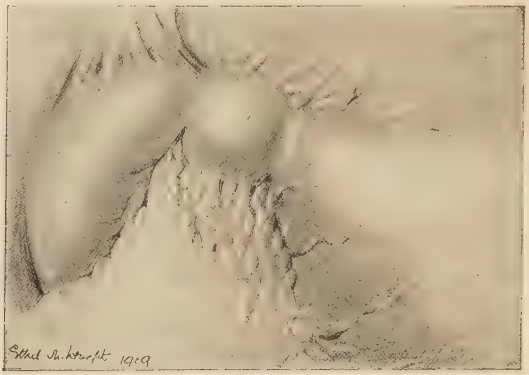

Fig. 261.—Double stenosis in the duodenum ("hour-glass duodenum") (Moynihan, after W. J. Mayo).

**Ulcus Carcinomatosum.**—Change from a simple ulcer of the duodenum to a malignant condition is extremely rare. W. J. Mayo[1] reports 4 cases. Perry and Shaw report 5 cases; Boxwell,[2] 1 case; Ewald,[3] 1 case; Eichhorst,[4] 1 case; and Arnold and S. Fenwick, each 1 case.

Cancer developing on the edge of a duodenal ulcer which has involved the stomach at the pyloric ring is more frequent.

**Clinical Aspects.**—Occasionally the cicatrix of an old ulcer may be found at autopsy, though there have been no symptoms during life. Other patients may be apparently in perfect health, when sud-

[1] Jour. Amer. Med. Assoc., 1908, ii, 558.

[2] Lancet, 1907, ii, 1687.

[3] Berl. klin. Wochens., 1886, No. 32, p. 527.

[4] Zeitschr. f. klin. Med., 1888, xiv, 519.

denly a severe and dangerous intestinal hemorrhage takes place, or perforation of the intestine and resulting peritonitis. As in *many cases the ulcer* is free from adhesions, there is great danger when perforation occurs. In some the *omentum* fortunately adheres.

**Symptoms.**—In discussing the symptoms of duodenal ulcer, the author must take exception to the statement of Moynihan that either hyperchlorhydria or acid gastritis " is " duodenal ulcer. Acid gastritis is a definite clinical entity; while hyperchlorhydria, in many cases, is a functional disturbance and readily cured. The writer does believe, however, that a " persistent hyperchlorhydria," or persistent symptoms simulating such, are suggestive of either gastric or duodenal ulcer. The determination of pus in the gastric contents alone, or with the presence of blood or occult blood, point to the stomach; while the absence of *pus in* the gastric contents, and the presence of blood or occult blood in the stool and sometimes in the stomach in addition, point to the duodenum. In some cases both stomach and duodenum may be involved.

**Acute Duodenal Ulcer.**—The writer has seen a few cases of acute duodenal ulcer of apparently brief duration. They occurred in males. There were pain and tenderness in the epigastrium to the right of the linea alba, over the duodenum; vomiting was rare, melena and collapse were marked, and subsequent gastric analysis showed *marked* hyperchlorhydria; no pus, no occult blood in the gastric contents. *As a rule,* duodenal ulcer is characterized by its *chronicity.*

The writer believes the anamnesis to be of exceptional importance, but that repeated and persistent tests for occult blood in the stool in suspected cases, even in the early stage, to be of diagnostic value, and also the use of the microscope, in that the latter will show the presence or absence of pus and aid in differential diagnosis. There are, however, certain symptoms which are characteristic of duodenal ulcer. Thus: A long history (chronicity); the periodicity of the symptoms, and their recurrence from time to time in " attacks "; and their complete abeyance in the intervals. The fact that persistent treatment fails to cure the patient is most suggestive.

The patient frequently dates his complaint from an early period, with intermitting periods of comfort. As a rule, he is from twenty-five to forty-five years of age; males are more frequently affected.

The early symptoms are a sense of weight, oppression, or distention in the epigastrium after meals. The discomfort appears usually two hours or a little more after food has been taken. There is comfort immediately after a meal; if pain or discomfort were present before, the meal relieves them for a time. Then, again, the pain is felt in two, three, four, or even six hours later. When it occurs three or four hours after food, Moynihan finds that the ulcer is " tucked back." And when the pain comes on earlier than two hours after food, either there are recent adhesions between the ulcer and the liver or abdominal wall, or pyloric stenosis is beginning to develop.

**Pain.**—Usually the pain comes on gradually and increases gradually in severity, being accompanied by a sense of fulness and distention ("a blown-up feeling"). There is later an eructation of fluid or gas, which affords relief. The interval between the taking of food and the onset of the pain is constant from day to day, providing the quantity and character of the food remain the same. With liquid food the pain comes earlier, as a rule; while with food that is solid and indigestible, the pain comes later; while with an ordinary meal (liquid and solid), the pain rarely appears in less than two hours. The author finds, however, that this is not invariably the case, as in a recent case examined, of seventeen years' duration, no solid food could be taken at all, liquids alone being ingested with comfort. Discomfort and pain appeared at the end of three hours, and this was temporarily relieved by milk feeding. "Hunger pain" has been suggested as descriptive of this symptom, since patients state that it appears when they are beginning to feel hungry. There have been several explanations offered as to the cause of hunger pain, and it would seem difficult to determine why bland liquid often causes an earlier appearance of pain. It has been clearly demonstrated by the *x*-rays *that liquids pass out along the lesser curvature early and with great rapidity.* The writer believes the earlier appearance of pain to be due to the earlier and more active pyloric contraction, with resulting movements of the irritable ulcer. Cannon has shown that the waves (motor) in the fundus are slight, and, unquestionably, for at least an hour a considerable portion of the solid food lies in this region, while it is further acted on by the ptyalin. The smaller quantities of solid food which pass from the stomach at this period are well diluted with liquid. Later, the undiluted chyme passes out in larger quantity, with increased pyloric movements, and also increased motility in the ulcerated region. Moynihan holds that probably the acid content is greater at that period, or possibly some other change has taken place in the chyme, which increases the irritating effects. Codman also calls attention to the primary symptoms of "hunger pain" and indigestion. The pain is, as a rule, noticed at first only after the heaviest meal of the day. For example, if dinner is taken between 1 and 2 P. M., the pain will appear regularly about 4 P. M. Frequently this is the only time when discomfort is felt; but later, or in subsequent attacks, the pain comes on at the usual interval after every meal; though temporarily, after the meal the pain is relieved, but only to return in time. The patient usually states that "food makes the pain better, and that the pain comes on when he begins to feel hungry." It is quite characteristic that the pain should awake the patient in the night, generally about 2 A. M. The patient soon realizes that food relieves the pain, and so has at hand a biscuit, or bread and butter, or milk, to be taken when it begins; and food is often kept beside the bed, to be taken early in the morning when awakened by the pain. The appearance of the pain at definite intervals after taking food is consistent. A sensation of weight, or fulness, or distention, in the epigastrium, often precedes or accompanies the pain. It may be of a boring, gnawing, or burning character, and it may be relieved by belching. The patient often endeavors to eructate gas in order to secure temporary relief. Slight regurgitation of food sometimes occurs. This may taste bitter or acid, and the throat may feel scalded, and the teeth like chalk. The salivary flow may be copious and cause considerable annoyance. In some, swallowing the saliva may temporarily relieve the discomfort. The author has seen some cases in which the pain was not marked, but there was more a sensation of dull ache or local discomfort.

For a long period, sometimes throughout the entire history, the pain may remain confined to the epigastrium. It may pass through to the back, or around the right side, and may radiate down into the abdominal cavity. In my own experience I have not seen it pass into the back, as in gastric ulcer, but it lies rather to the right of the linea alba. Pressure at times gives relief, such as by hugging a pillow to the abdomen. In some cases the pain is cramp-like, a spasm is felt with exacerbations and remissions, as in colic. This is probably due to spasm of the pylorus. Great epigastric distention occurs with it, with a feeling almost of bursting. The appetite remains good during the period of pain.

The patient may eat less than he desires to, since he has learned by experience that excess or even satiation of the appetite may be followed by a more enduring pain.

Fluid food, as previously noted, when taken alone, may cause the pain to come earlier and to last longer than after ordinary meals. For a while the patient *may feel worse on the liquid diet;* but *persistence in the latter will bring relief after a time.*

Vomiting is infrequent and is rarely present until stenosis develops.

Stenosis appears in the late period when the ulcer is healed or nearly so.

Pyloric spasm may produce an evanescent stenosis. The symptoms are *periodic*, they recur from *time to time in "attacks"* and they are in *abeyance in the intervals.* The "attack" may come on as a result of exposure to cold, wetting the feet, a hasty or indigestible meal, or worry or overwork.

"Catching cold" is a frequent cause, the attacks being most prevalent in December, January, or February.

The symptoms are nearly *always absent in summer.*

The attack may follow another disease, such as influenza. The attacks may vary in length from two to three weeks to several months, and a few days rest in the country or at the seashore may cut them short.

The onset and termination may be sudden. A chill may bring on the attack immediately and the symptoms may continue for months. At times they may cease abruptly. On the other hand, the pain may become a daily occurrence and retain its peculiar character.

In the intervals between attacks there is complete immunity from pain, and food is taken with enjoyment, there is no discomfort, and there is a gain in weight. The recovery may apparently be complete. The patient or physician may believe the case to be one of acid dyspepsia or a neurosis.

All these symptoms may be present for years *without any physical signs.*

Though the anamnesis is extremely important, the writer does not agree with Moynihan's statement, p. 115, "Duodenal Ulcer," [1] that it is not necessary for an accurate diagnosis that any examination of the patient should be made." Repeated and frequent examinations of the stool should be made for occult blood. The writer believes *it can usually be discovered.* Bleeding from hemorrhoids should, of course, be excluded. The *test-meal* is also *important.*

In many cases, especially in the stage when the ulcer is latent, or between the presence of symptoms, nothing may be revealed by physical examination. There may be some epigastric tenderness in the middle line or to the right. During the period of pain, between two or three hours after a meal, there is usually some tenderness, which may rarely be marked. This may be in the middle line over an area of 2 or 3 inches. The pyloric region may in some be sensitive. Tenderness on pressure over a small circumscribed point is more frequent with gastric ulcer

In some cases, however, of duodenal ulcer, there may be continuous pain and tenderness due to a local peritonitis. Rarely pain and tenderness appear on the left side for which there is no explanation. When acute pain is present the right epigastric reflex may be strongly accentuated. Local tenderness is usually combined with local rigidity, and the upper part of the right rectus muscle may be contracted and tense.

Hence, tenderness in the middle line and to the right, accentuation of the right epigastric reflex, and rigidity of the right upper rectus are the chief physical signs of duodenal ulcer; they may be absent.

Vomiting is rare with duodenal ulcer, unless there is stenosis with dilatation of the stomach, and this condition occurs in the later stages. With reflex spasm of the pylorus, vomiting occasionally occurs, but it does not relieve the pain of duodenal ulcer as it does that of gastric ulcer. A regurgitation of acid chyme is, however, quite frequently present.

When stenosis occurs, we have the physical signs of dilatation of the stomach, with peristaltic unrest, vomiting, motor insufficiency, etc.

S. Kemp [1] believes that hypersecretion suggests either gastric or duodenal ulcer as a rule, and when it is excessive, it speaks strongly for duodenal ulcer; in fact, he believes it more frequent with the last. Pyloric spasm was quite frequent with duodenal ulcer, and gastric motor insufficiency he believed to be the rule. The author only finds hypersecretion, and motor insufficiency in the advanced cases with stenosis.

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[1] Second edition.

[2] Ungekrift for Laeyer, Copenhagen, Dec. 15, 1910, lxxii, Nos. 49 and 50.

*Bowels.*—Constipation is the rule, diarrhea rarely occurs.

*Gastric Contents.*—The test-breakfast should be given in all cases. The patients usually complain of an acid stomach (hyperacidity). In the writer's own experience, in the cases with acute symptoms, with melena or a clear history of hemorrhage, he has found hyperchlorhydria present. This corresponds with Boas' observations. On the other hand, the intractible cases of so-called " acid dyspepsia " of long standing which do not yield to remedies, and which are recurrent, Moynihan believes to be due to duodenal ulcer. In the type of chronic duodenal ulcer, the gastric juice usually contains less free HCl than normal, in spite of the symptoms of hyperchlorhydria. On the other hand, the cases which have progressed to stenosis and dilatation of the stomach have hyperacidity as a rule; but in these the chief symptoms are referable to the *mechanical obstruction.* Even the absence of free HCl and the presence of lactic acid have been reported in cases of duodenal ulceration.

As about 50 per cent. of one's private office cases suffer from true hyperchlorhydria (an excess of free HCl above normal, in addition to excessive total acidity), the writer absolutely disagrees with Moynihan in his statement that the term hyperchlorhydria is a misnomer, is due to an organic condition, and is never functional, and that the presence of an excess of acid is most infrequent. Hyperchlorhydria results chiefly from dietary indiscretions, and also as associated with chlorosis, gastroptosis, and nervous conditions. There may be attacks with free intervals, just as in the case of duodenal ulcer; excepting in the pure nervous cases, or those reflex from the gall-bladder and appendix, the character of the food may influence the attacks.

Simple hyperchlorhydria is, as a rule, amenable to treatment. Hyperchlorhydria with gastric ulcer is present in the acute cases—in the author's opinion, with a history of hematemesis and melena, or in apparent hyperacidity cases not amenable to treatment. Hyperchlorhydria with duodenal ulcer occurs in acute cases with melena or a history of the same.

*Symptomatic hyperacidity—recurrent and intractable with* less *HCl than normal is suggestive of duodenal ulcer.*

The presence of blood visible or occult, and pus in the gastric contents, are diagnostic of gastric ulcer. Of course, pus from the pharynx or from gastric abscess must be excluded. Preliminary antiseptic nasopharyngeal sprays, a preliminary lavage, and sterile diet are valuable methods to exclude ingested pus.

The determination of pus, therefore, diagnoses ulcer affecting the stomach either benign or malignant in type, or achlorhydria hæmorrhagica gastrica. In the latter condition other peculiarities of the gastric contents are present which are described under that subject. With achlorhydrin hemorrhagica free $HCl$ notably is *absent*, and micro-organisms are in excess.

On the other hand, melena is most frequent with duodenal ulcer and hematemesis is less frequent.

If the examination of the gastric contents show *an absence of pus*, occult blood present or absent, and occult blood [1] be found *present* in the stool, the case is one of duodenal ulcer.

Acid gastritis is hyperchlorhydria with mucus, an early stage of chronic gastritis and an *entity in itself*.

These features show the importance of the test-meal. Gross has recently suggested aspirating the duodenal contents with his duodenal pump and examining for blood and occult blood. The method may possibly prove of service.

*Hemorrhage.*—This sign sometimes appears early, but is more frequently a late symptom. Visible hemorrhage Moynihan believes a sign of neglected opportunity. Various authors place the frequency of hemorrhage as occurring in about one-third of all cases, while Moynihan notes it in 37.6 per cent.

When hemorrhage occurs, it may manifest itself either as hematemesis vomiting of blood or as melena (blood in the feces); as a rule, the blood has a tar-like or coffee ground appearance. Sometimes both occur, but melena *is the most frequent*, without hematemesis.

Hemorrhage from duodenal ulcer is far more serious than bleeding from a gastric ulcer. There may be first an exacerbation of the indigestion and a feeling of distention. The patient may suddenly become faint and weak, the head feels light, and he becomes pale, the forehead covered with sweat, and he asks for air. There are all the signs of an internal hemorrhage. Tarry blood may be voided in the stool or brighter blood vomited. The patient may bleed to death without any visible blood. Death from hemorrhage may be nearly instantaneous, when such vessels as the aorta, the hepatic, superior pancreaticoduodenal right gastro-epiploic and pyloric arteries, or the portal or superior mesenteric veins are eroded.

On the other hand, there may be progressive anemia and weakness with no particular symptoms, resulting from smaller repeated hemorrhages.

Undoubtedly careful and repeated examination of the stools and gastric contents for occult blood by Weber's, the benzidin, or phenolphthalein tests, would demonstrate that ulcer is present in every suspected case. A number of examinations may be necessary, but the writer believes *occult blood* can ultimately always *be found in the stool*. A meat-free diet for two days, administration of a *coarse* diet, and elimination of bleeding from hemorrhoids, are necessary precautions. Celery, radishes, etc. (course ballast), fret the ulcerated surface and tend to cause slight hemorrhage.

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[1] Hemorrhoids and other causes must be excluded.

Einhorn suggests the thread impregnation test, as described under the gastric ulcer. The writer believes, however, that if blood is present in sufficient quantity to discolor the thread, the occult blood test will invariably be present. The thread test is uncertain.

Moynihan's statistics are as follows: 70 patients (37.6 per cent.) gave a history of bleeding. Of these 7 had hematemesis alone, 23 had melena alone, 30 had both hematemesis and melena.

Out of 139 cases in which duodenal ulcer alone was found, hemorrhage was noted in 49 cases (35.2 per cent.); 9 had hematemesis alone (6.4 per cent.); 18 had melena alone (13 per cent.); 22 had hematemesis and melena (15.8 per cent.). In 18 cases the hemorrhage was severe—in all 9.2 per cent. suffered from dangerous bleeding.

*Jaundice.*—Jaundice is rare with duodenal ulcer. The development of icterus in a case presenting some of the symptoms of gastric ulcer, probably shows duodenal ulcer, if gall-stones can be excluded.

The death-rate from hemorrhage is quite large and is variously estimated at from 13 to 33 per cent.

*Tetany.*—Three cases gave a history of tetany.

*Cardiospasm.*—In 3 cases cardiospasm was present.

*Dilatation of the Stomach.*—In a small number of cases the symptoms of active ulceration are latent and the physician is first consulted for copious and repeated vomiting. The stomach is dilated—there is peristaltic unrest—the symptoms are those of *pyloric stenosis.* The history may possibly differentiate between gastric and duodenal ulcer—but the author believes this result can only be positively attained by operation, which he holds is *invariably imperative* in such cases. In this type the ulcer is usually nearly healed, and the cicatricial contraction is responsible for the stenosis.

*Perforation.*—Perforation is the most serious complication that can occur with duodenal ulcer. It may take place in acute or chronic ulcer, both in the young and old.

Finney[1] reports a case in a child two months old, while Moynihan has seen it in a woman aged seventy-seven.

Sudden perforation may occur with the acute duodenal ulcer, due to burns, septicemia, etc.

Perforation may be acute, subacute, or chronic. In Moynihan's series of 11 cases, acute perforation occurred in a chronic ulcer.

In every case, there was evidence that the ulcer had long been present; but only recently was the ulcer the seat of a more active pathological change, and there had been an exacerbation in the symptoms. Occasionally a perforation may occur without previous symptoms.

*Symptoms of Acute Perforation.*—There is a sudden onset of intolerable and agonizing pain. This is so severe, that sudden death may occur from it. The patient is prostrated, he is pale and faint, the expression anxious, and sweat appears on the brow. Respirations are short and quick. Deep inspiration causes a groan of agony and a spasm of pain. Replies to questions are given in snatches and expiration ends abruptly in a catch. Collapse may be complete in some cases, the author finds in his own experience. In some, however, it may not be apparent. When the patient is seen an hour or two after perforation, at which time the pulse may not be particularly rapid, *muscular rigidity* is a marked symptom and the abdomen is at this time retracted. Distention comes later. The *tenseness of the abdominal muscles* is persistent. On overcoming the *protective muscular rigidity*, abdominal tenderness is present. There is an area of more *marked tenderneess* and *rigidity to the right of the median line above the umbilicus.*

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[1] London Lancet, 1908, ii, 1748.

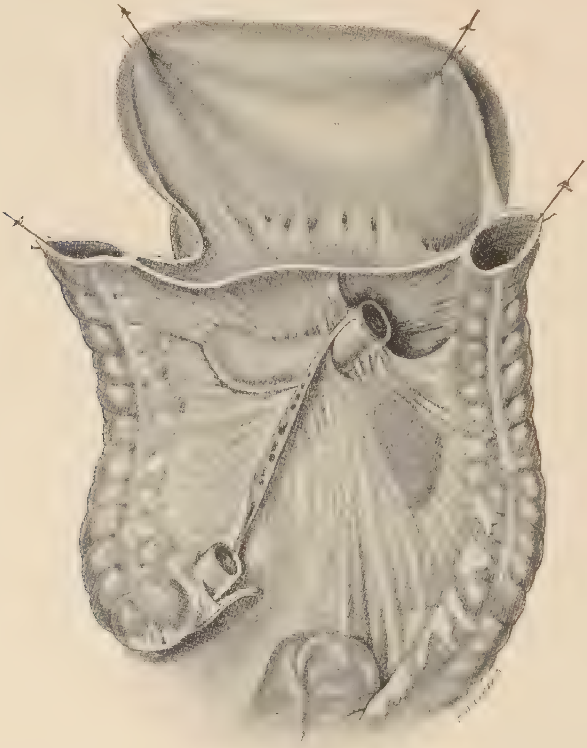

Fig. 262.—Showing the disposition of the duodenal peritoneal coat, and explains the direction taken by duodenal contents in case of perforation (Gaultier).

Vomiting may occur at first, but it usually does not.

The liver dulness is not yet impaired. Percussion of any part of the abdomen is resented by the patient.

As a rule, the symptoms rapidly alter as fluid escapes through the rupture into the peritoneal cavity, producing the symptoms of acute peritonitis.

The pulse-rate rises steadily, the quality becoming poorer. The abdomen, preserving its rigidity, becomes fuller, until a uniform tight distention occurs. Tenderness, as a rule, becomes more marked on the right side, and the right iliac fossa becomes the most tender and most distended region. Leukocytosis and increase of polynuclears are present.

The temperature, which was previously normal or subnormal, begins to rise and may reach 102° F. or more. Intestinal stasis is absolute from the first. Repeated enemata may bring a little gas, and feces in some cases. Interference with respiration persists and increases as the abdomen distends. The face becomes livid, the face and hands cold and damp, and, finally, cyanosis develops. From the time of perforation to the death of the patient a period averaging from two to five days may elapse.

The right side of the abdomen, as heretofore noted, is chiefly affected, so that the mistaken diagnosis of perforative appendicits has been frequently made. The fluid escaping from the duodenum is prevented from passing to the left by the reflection of the great omentum from the duodenum and the stomach. It flows down to the outer side of the colon to the right iliac fossa and brim of the pelvis and then overflows into the latter. If the ascending colon is bound down and has no mesentery, the fluid may rise alongside the colon, crossing a few inches below the hepatic flexure, and then flow to the appendix region, guided by the obliquity of the mesentery (Figs. 262 and 263).

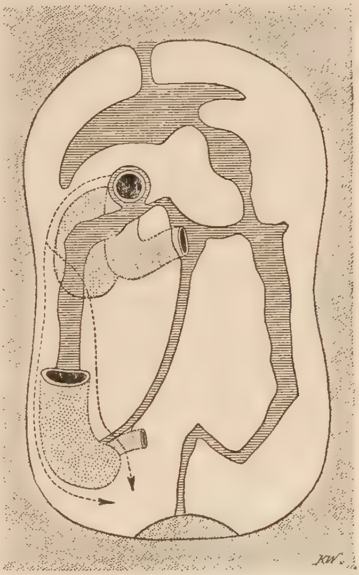

Fig. 263.—Schema, after Moynihan, showing direction taken by duodenal contents in perforation of the duodenum; how they arrive in the region of the cecum and simulate appendicitis.

**Differential Diagnosis between Acute Perforation of Duodenal Ulcer and other Conditions.**—*Duodenal Ulcer.*—The previous history of the case is of great value; it occurs more frequently in men. Pains appear two hours or later after meals. At the time of perforation the pain is *overwhelming in character.* It is frequently to the right of the median line in the epigastrium, though it may be referred to the whole abdomen. Within one to two hours, however, the pain is always of greater severity on the right side, and tenderness and muscular rigidity are also excessive there. The rigidity is extreme. No conditions, except perforation of the duodenum or stomach, give rise to such extreme tension in the muscles. Rigidity also persists, and retraction of the abdomen continues until distention due to peritonitis develops. Rigidity does not lessen until profound toxemia ensues.

*Gastric Ulcer.—Perforation.*—Pain appears earlier; frequently local tenderness, dorsal pain, and other symptoms precede the perforation, though not always so. With perforation of the stomach, the pain is usually to the left of the median line. After perforation, careful examination will show an area which is more resistant and more tender, beneath which the ulcer lies, and hence, the site of the perforation. When the most tender area is in the left hypochondrium, the perforation is toward the cardia; if around the umbilicus, it is in the body of the stomach; and if to the right of the median line or in the right hypochondrium, then the perforation is probably in the pyloric end of the stomach or in the duodenum.

*Perforative Appendicitis.*—The pain may be first referred to the epigastrium or to the entire abdomen, and then become localized in the right iliac fossa. There may be a dyspeptic history, but not the typical history of gastric dyspepsia, as with duodenal ulcer. There is generally a history of preceding pain in the appendical region, or of previous attacks, or of constipation, or colitis. In many cases an aperient has been taken previous to perforation. The muscular rigidity with appendicitis is not as intense as with duodenal perforation, nor are the pain and agony as intolerable. The diaphragm is not held as tight and the respirations are not as short and jerky.

In both conditions there is tenderness in the right iliac fossa. Tenderness and rigidity are always present in the right hypochondrium with duodenal perforation, and shortly after perforation of the duodenal ulcer, rigidity of the right rectus (upper part) and tenderness are most marked in this region.

With appendicitis they are exceptionally there, and only when the appendix is retroverted and extends to the liver. Even then the tenderness is less marked. Rigidity of the lower portion of the right rectus is usual.

Moreover, the test *Head's zone for the appendix* is of value in these cases, as it lies below the umbilicus. Rarely perforation of ulcer and perforative appendicitis may coexist.[1]

*Acute Pancreatitis.*—As a rule, there are no initial symptoms. Occasionally acute pancreatitis may be dependent on the impaction of a stone in the ampulla of Vater, in which event there may be the previous history of "gall-stones" or "gall-stone dyspepsia." The pain of acute *pancreatitis is more in the epigastrium.* Muscular rigidity, tenderness, and distention chiefly occur there—"an epigastric peritonitis"—and the enlarged and edematous pancreas may at times be felt on deep palpation. Fitz describes scattered areas of abdominal tenderness lying probably over the areas of fat necrosis.

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[1] B. Carter, London Lancet, 1901, ii, 1195.

The pulse is bad from the start, and it is *always rapid* and of poor quality. Its rate is at first disproportionate to the severity of the other symptoms. Constipation is marked. Vomiting is more frequent. It consists of food, bile, and black altered blood. Blood is frequently passed from the intestines. A deep lividity or cyanosis of the skin, *chiefly of the face*, occurs. Corpulent people are often attacked by acute pancreatitis, and it occurs with considerable frequency during the early months of pregnancy.

With acute pancreatitis, a general distention of the abdomen is rare, the right side of the abdomen is not specially involved, and the hurried, jerky respiration due to contraction of the diaphragm does not occur. Fat necrosis is present with acute pancreatitis. A case of fat necrosis with peritonitis resulting from perforation of a duodenal ulcer is reported by Richter[1] as a rare occurrence. The pancreas was found to be normal.

*Acute Perforation of the Gall-bladder.*—The local and general symptoms may simulate acute perforation of duodenal ulcer.

A careful investigation into the previous history is the only means of diagnosis. A history of hepatic colic, with occasional attacks of jaundice, can usually be secured. Moreover, the illness does not seem as desperate, nor is the attack of acute pain as severe, as when the duodenum perforates.

*Acute Thoracic Disease.*—The mimicry of appendicitis is more usual than that of perforation of ulcer of the duodenum.

During the onset of pneumonia, diaphragmatic pleurisy or acute bronchitis, the pain may be felt exclusively in the abdomen, the abdominal muscles may be tense and the surface tender. There are several features which are an aid to differential diagnosis:

(1) The temperature in acute lesions of the thorax often ranges higher—103° to 104° F.—than it does in acute abdominal lesions (rarely above 102° F.).

(2) The respirations are more rapid—35 to 40 or more—in pulmonary conditions.

(3) There is a disproportion in the early stages in thoracic lesions, between the pulse and respiration. The pulse may not be much over 100, and the respirations 35 to 45. A more rapid pulse-rate, 120 or more, with a slower respiration, about 25, would be most frequent in an abdominal lesion.

(4) In abdominal conditions there is persistent rigidity and deeper tenderness. With thoracic inflammation the tenderness is superficial, and deep pressure may even give relief, and during respiration, at the height of expiration, the abdominal muscles may be soft and the rigidity absent. The rigidity is less marked and less persistent and the area affected is limited.

(5) With diaphragmatic pleurisy, the respiratory movements in the upper thorax are exaggerated, while they are slower below.

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[1] Quarterly Bulletin Northwestern University Medical School, Dec., 1910.

(6) Head's zones of cutaneous hyperalgesia for the lungs lie over the thorax, while for the duodenum Head's zone lies just below the free border of the ribs, chiefly to the right of the median line, and for the appendix, it lies below the level of the umbilicus.

**Treatment of Acute Perforation of the Duodenal Ulcer.**—In brief, immediate operation is indicated; suture of perforation, drainage, usually gastro-enterostomy. Subsequent to operation, proctoclysis in Fowler's position is most valuable. Resection of the ulcer may be required.

**Subacute Perforation of Duodenal Ulcer.**—In this case, though there is a sudden rupture of an ulcer, as in the acute perforation, there is a definite localization of the fluid escaping through the rent and in many instances a narrow circumscribed peritonitis.

An empty stomach at the time of perforation would render the symptoms less severe. A sudden movement or violent strain may be the cause of the rupture. A tag of omentum may plug the opening of the ulcer, or peritoneal irritation, as the ulcer deepens, may cause plastic lymph deposits on the base of the ulcer, so that there is a nearly impenetrable barrier at the moment of perforation.

Moreover, the duodenum may become adherent at the base of the ulcer to the anterior abdominal wall, the liver or the pancreas. The symptoms are the same as in acute perforation, except that they are *less intense.*

There is a previous history pointing to duodenal ulcer; in some cases there is increased pain for a few days previous to perforation, or the abdomen is stiff and sore, and discomfort is felt on laughing or stretching.

The pain of perforation is again sudden in its onset and severe in character, but not as intolerable as in acute perforation. Vomiting and prostration may follow. The abdomen is everywhere tender; but there may be a particularly tender and resistant area, which on palpation may feel like a flat, hard mass inserted in the wall of the abdomen. Leukocytosis and increased polyneuclears are present.

The symptoms subside slowly, the character of the pulse improves, vomiting ceases and the abdomen, which was at first hard and retracted, may become soft, except at the one spot, or it may be slightly distended, with some free fluid.

If no operation is undertaken, there may be several results, either a periduodenal abscess may form, or a secondary rupture into the general peritoneum may occur, or the adhesion of the ulcer to the abdominal wall, liver or pancreas may increase in firmness, the acute inflammation subside and the patient live for years. This last is more common.

*Differential Diagnosis of Subacute Perforation of the Duodenum.*—The chief difficulty is differentiating this condition from acute cholecystitis. In both conditions there are pain, sudden in onset, which is severe and often colicky. There is no general invasion of the peritoneum, but a localized peritonitis with a tender resistant area. The best clue is the previous history of the case. The determination of Head's zone may be of service.

*Treatment.*—Since, in the early stages, a differential diagnosis between acute and subacute perforation cannot be made, in view of the uncertainty, immediate operation is indicated.

**Chronic Perforation of Duodenal Ulcer.**—In this type of perforation the intestinal coat is destroyed slowly by the ulcer, and by the time the serous coat is reached a protective barrier, often reinforced by omentum, is raised by newly formed lymph deposit and adhesions, so that the inflammatory process is circumscribed and perforation into the general peritoneal cavity does not occur, but a local peritontis.

The local cavity increases in size and the contents may become purulent, so that a periduodenal abscess results.

In other cases, the perforation may occur suddenly, but be so minute in size or the duodenum be so empty that leakage is excessively small. In this event, the inflammation may become rapidly circumscribed by adhesions and again a periduodenal abscess result. Communication with the duodenum may then be shut off completely, or it may be closed in such a way as to reopen when the abscess has been opened. A duodenal fistula then results.

The periduodenal abscess may perforate, or burrow in various directions. Fistula may be present between the duodenum and the gall-bladder[1] or between the duodenum and colon[2] or hepatic abscess[3] may result; or a pancreatic[4] duodenal fistula.

A few cases are also recorded of fistula between the stomach and duodenum; but in these cases it seems probable the primary ulcer was in the stomach. Subdiaphragmatic abscess may result from chronic perforation of duodenal ulcer.

A periduodenal abscess may burrow to a great distance; thus it may pass into the retroperitoneal spaces and ascend along the greater vessels into the neck, or may point between the seventh and eighth ribs posteriorly, or at the tip of the right scapula. It has passed down along the ascending colon to the right iliac fossa and burst into the cecum or may lead to an abscess of the right iliac fossa and pelvis. It has burrowed beneath the right psoas sheath, causing flexion of hip and lordosis. It may penetrate the gall-bladder.

Meunier[5] reports a case where an abscess cavity was found on the under surface of the liver. It was transversed by the duodenum, which was completely severed by a circular ulcer.

The abscess which forms may reach the anterior abdominal wall and perforate the same with resulting external duodenal fistula.[6]

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[1] London Lancet, 1850, i, 776; Schmidt's Jahrbuch, cxxxix, 293.
[2] Pathol. Soc. Trans., 1862, xiv, 173.
[3] Munch. med. Wochens., 1887, i, 678.
[4] Guy's Hosp. Museum, No. 757.
[5] Bull. Soc. Anat., 1893, lxviii, 487.
[6] Archiv. Gen. de Med., 1887, i, 414 *et seq.;* also Bull. Soc. Anat., 1870, xv, 429.

These cases ultimately die of inanition and sepsis unless definite surgical procedures are adopted.

*Treatment.*—Appropriate surgical measures are indicated for chronic perforation of a duodenal ulcer.

**Differential Diagnosis of Duodenal Ulcer.**—This matter is so important that it is deemed proper to recapitulate some former observations.

**Duodenal Ulcer.**—This condition is more frequent in males; pain after food does not appear until two hours or more; the hunger pain is relieved by food; pain may be in the median line, though quite frequently it is to the right of it, and may radiate over the right costal margin to the breast or round to the back. Tenderness on pressure, when present, is always to the right of the median line. It is, as a rule, not as circumscribed as with gastric ulcer. Attacks are apt to be recurrent in the cold and wet seasons. Vomiting is rare, unless there is stenosis of the duodenum or pylorus. Melena more usually occurs without hematemesis; hemorrhage is more dangerous; perforation more frequent; latent type with sudden hemorrhages is more frequent; history is usually chronic, symptoms recur in attacks; patients complain of hyperacidity. Gastric findings, usually in chronic cases, show no excessive content of HCl, in fact, as a rule, it is less than normal. On the other hand, in the cases with brief history and *acute hemorrhage*, I have generally found hyperchlorhydria. These last are of easy diagnosis. In the more obscure cases repeated examinations of the stool for occult blood *will invariably show its presence.* The gastric contents *show no pus*, and at times blood or *occult blood* is present. The presence of pus *shows an ulceration in the pylorus* or stomach. The determination of *occult blood in the stool*, in cases where there is no definite history of hemorrhage or visible evidences of the *same, is of great importance.* In an obscure case at the Red Cross Hospital, a positive diagnosis of duodenal ulcer was made chiefly on this point and was confirmed at operation by Herman Haubold. It served to exclude gall-bladder disease and a gastric neurosis. The special Head zone aids differentiation of ulcer of the duodenum from disease of the gall-bladder and stomach.

*Gastric Ulcer.*—This is more frequent in women, is frequent with chlorosis; pain is earlier, within a short time or less than an hour after food, rarely within one to two hours; pain in the median line or to the left and may radiate to the left margin, left breast, and even down the left arm; dorsal pain is present at a point between the scapula and spine; tenderness is more circumscribed and is in the median line and often to the left of the same; there is not the same dependence of the attacks on the climate or seasons; vomiting is more frequent; hematemesis more frequent; melena less so, but quite often present; hyperchlorhydria is present in the acute cases; diminished acidity or absence of HCl may occur in the chronic ulcers.

*Pus always present in the gastric contents; occult blood* or blood is found in the gastric contents. *Pus is extremely important,* as in a case of Connor's, the diagnosis was made from its presence alone, no occult blood being found. Occult blood in the stool is frequent. No mucus is present in the gastric contents.

Bassler holds that 2 per cent. or more of gas in hyperchlorhydria is suspicious of ulcer.

*Simple Hyperchlorhydria.*—This is a very frequent condition—*content of HCl is always high and constitutes chief acidity*—total acidity is high. No pus in *gastric contents and no blood or occult blood.* No occult blood is found in the stool. There is no special predilection for either sex. Dietary indiscretions, anemia, gastroptosis, nervous conditions, etc., may be the cause. Cases may be persistent, but are amenable to relief by treatment. When hyperchlorhydria occurs with other conditions, such as appendicitis, gall-bladder disease, and gastric or duodenal ulcer, the anamnesis and physical examination for the first two conditions and the presence of gastric and intestinal occult blood with the last two will aid diagnosis. With the ulcerated conditions, gall-bladder disease, and appendicitis special Head zones are present. There is no mucus with simple hyperchlorhydria.

*Acid Gastritis.*—There is hyperacidity plus mucus. It is an initial stage of chronic gastritis.

*Achlorhydria Hæmorrhagica Gastrica.*—There are absence of free HCl, *presence of mucus,* many actively growing bacteria, pus, blood, or occult blood. The condition is reflex. Gall-bladder disease or appendicitis are a frequent cause and the condition is relieved by operation.

*Cholelithiasis.*—There are no definite attacks which occur at certain seasons (no periodicity); the pain is unendurable; is more severe than duodenal ulcer, except when there is perforation, which last has associated definite symptoms. Pain occurs within an hour after food. There are often a " catch in the breath," depression, nausea, and sweating. The pain begins *suddenly,* is *abrupt in the onset* and in its relief, as it may rapidly pass away; acidity or heartburn may be present.

Food or an alkali does not relieve the pain and the idea of food is repugnant. Chills, sweats, a " feeling of gooseflesh," shivering, and subsequent heat are present. There may be a sensation of pain in the shoulder-blade, which is suggestive of gall-stone impaction in the cystic duct. The pain is cramp-like, passes through and around the right side to the shoulder-blade, and epigastric distention accompanies it. A feeling of stiffness or soreness remains for some hours. The gall-bladder is tender and Head's zone is present.

*Ulceration of a Gall-stone into the Duodenum.*—The writer has seen a case suffering from an attack of acute pain to the right of the median line in the epigastric region, followed by several attacks of hematemesis and marked melena. One consultant believed it due to hemorrhage from an occult duodenal ulcer. The patient was a stout, elderly woman, with an apparent history of previous gall-stone attacks. The possibility of subacute pancreatitis with hemorrhage was suggested. Subsequently a gall-stone the size of an English walnut was removed from the intestines. It had ulcerated through from the gall-bladder, eroding a large vessel. The patient recovered.

*Acute Pancreatitis with Gastro-intestinal Hemorrhage.*—As a rule, there is a history of previous attacks of gall-stones. There is an "epigastric peritonitis," and the attack is fulminating. See Acute Hemorrhagic Pancreatitis.

*Chronic Pancreatitis, Simulating Gastric or Duodenal Ulcer, with Sypmtoms of Pyloric Stenosis, Moderate Dilatation of Stomach.*—This condition is particularly interesting. The writer recently examined a case (female) for John Connors presenting the following symptoms: pain one to three hours after eating; tenderness in the epigastrium; occasional vomiting; history of apparent coffee grounds; complains of hyperacidity, but acidity and HCl within normal limits, three years' history; stomach dilated to umbilicus; no pus; no occult blood found in gastric contents; no occult blood in stool (two examinations). The writer found evidences of mechanical obstruction to the exit of gastric contents. In view of absence of pus and occult blood in the same, believed obstruction to be on the duodenal side, and probably due to ulcer. Stool showed some disturbance of intestinal functions, but chronic pancreatitis was not suspected. Laparotomy showed no ulcer, *but mechanical obstruction to the duodenum* from *an enlarged* head of the pancreas; also a chronic pancreatitis, no obstruction to the common bile-duct, and no gall-stones. The common bile-duct evidently lay outside the enlarged head of the pancreas. The gall-bladder was opened and drained. It contained no gall-stones, but thick inspissated bile. This case is of particular interest. Whether subsequent to operation the enlarged head will subside or gastro-enterostomy will be necessary, it is too soon to determine. Patient is doing well.

*Banti's Disease.*—The anamnesis and the presence of the enlarged spleen are an important aid to diagnosis.

One should also determine that cirrhosis of the liver is not a factor in gastric intestinal hemorrhage and that the patient is not a "bleeder."

I have recently seen a most interesting case of persistent intestinal hemorrhage with indefinite gastro-intestinal symptoms of four years' duration, in which duodenal ulcer was believed to be the cause. Operation by Meeker at the Red Cross Hospital showed a single male lumbricoid worm 11 inches long in the jejunum—1 foot below the duodenum. There were evidences of numerous perforations of the intestinal mucosa, with resulting hemorrhages. No eosinophilia was present, and no other worms or ova were found. The worm was coiled into a U and was very active. This is the first such case I believe recorded of a lumbricoid worm producing intestinal hemorrhage, though it has occurred with ankylostoma.

**Prognosis.**—The prognosis is, as a rule, quite serious, especially in the cases with frequent recurrent hemorrhage. Perforation requires immediate surgical procedure. Relapses may occur in the apparently cured cases, but I have seen perfect recovery follow proper treatment.

**Treatment.**—This is similar to ulcer of the stomach.

*Hemorrhage.*—For acute hemorrhage, apply an ice-bag to the epigastrium and give at once a hypodermic of morphin, $\frac{1}{4}$ grain (0.016).

Trémolière's solution, calcium chlorid (2 per cent. solution) in gelatin (5 per cent. solution), 1 ounce (30.0) every two to three hours by mouth, is useful. Gelatin (5 to 10 per cent.), by mouth, 1 ounce (30.0), or a 2 per cent. solution subcutaneously may be employed; calcium chlorid or, preferably, calcium lactate, 10 grains (0.6) in 4 ounces (125.0) of water every three hours by mouth, or by enema if vomiting, or strontium or magnesium lactate by hypodermoclysis, 15 to 30 grains (1.0–2.0) in 4 ounces (125 c.c.) of sterile water, or ernutin, 5 minims (0.296) by hypodermic, are all useful.

Adrenalin (1 : 1000), 5 minims (0.296) or more, has been recommended by mouth or by hypodermic. It may too markedly raise pulse tension. Normal horse-serum or human serum by mouth or the latter, preferably by hypodermic, 15 to 30 c.c., might prove of value.

Spriggs[1] advocates the use of olive oil or almond oil, as suggested by Cohnheim and Walko. He gives $\frac{1}{2}$ to 1 ounce (30.0–60.0) t. i. d., increasing the dose to 2 ounces (120 c.c.) in some cases. Spriggs often starts the treatment by giving the olive oil alone by mouth every three hours, and nothing else except water for the thirst. This method is continued until the blood disappears from the stool. He then gives cream, and the foods of the Lenhartz diet are added, excluding rice. The oil is then reduced to 1 dram (4.0) before each meal.

*Author's Method.*—The author advocates the ice-bag, morphin by hypodermic, lactate of calcium, 10 grains every three hours in 4 ounces (125.0) of water, and 1 ounce (30.0) of a 5 to 10 per cent. gelatin solution every three hours; ernutin, 5 minims (0.296) by hypodermic, is useful, and in severe cases the serum treatment by mouth or hypodermic.

In a recent case with a tendency to vomit, the lactate of calcium was first given by rectum. After two doses it was not retained. Lactate of calcium, 15 grains (1.0), was then dissolved in 6 ounces (185 c.c.) of water and dram doses of this given every half hour, with similar doses of 5 per cent. gelatin solution, 1 dram (4.0) each. The result was most successful.

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[1] Brit. Med. Jour., May 21, 1910.

Olive oil, 1 ounce (30.0), in which bismuth subnitrate, 1 dram (4.0), is suspended, may aid in coating the bleeding surface. With a persistent hemorrhage, surgical procedure may be indicated, and it is certainly called for in persistent recurrent hemorrhages.

For *collapse,* rectal normal saline enemata at 120° F., a pint to a quart, proctoclysis, or hypodermoclysis are indicated. Rarely infusion may be required. In one case the writer needled a superficial vein and injected saline solution by this method. Camphorated oil, 5 grains (0.3) camphor in 20 drops of almond oil, repeated every hour for several doses, and strychnin sulphate, $\frac{1}{60}$ to $\frac{1}{30}$ grain every three hours, may be required. During the stage of collapse rectal feeding is indicated. White of egg and gelatin solution (cold) may be given by mouth. They take up free acid and the latter tends to check hemorrhage.

*Diet.*—Unless collapse be present, the author's modification of the Lenhartz method of diet the writer believes most efficacious, to be begun directly after hemorrhage, if it has occurred. This relieves best the hunger pain and takes up the free HCl. The patient should remain in bed and the general method of treatment followed as with gastric ulcer.

Hyperchlorhydria if present should be treated, and it is advisable, even if the HCl be less than normal but in fair amount, to administer an alkali, such as milk of magnesia, magnesia usta or soda bicarbonate, since the patient complains of acidity and evidently there is some irritant action from the acid.

Iron and arsenic should be given for the anemia caused by hemorrhage.

The bismuth treatment should be followed out, with which the silver nitrate may be alternated, as in gastric ulcer.

The *medical treatment should be tried* for the first, second, and, possibly, the third attack. I have given one patient recently the benefit of the doubt. He had a duodenal ulcer with two hemorrhages a year apart. I advised medical treatment for the present with careful watching, and a resort to surgery if further hemorrhage ensued.

An acute attack with marked hyperchlorhydria, as in the similar type of acute gastric ulcer, seems most amenable to medical treatment unless there be dangerous hemorrhage or perforation.

*Surgery.*—With the above exception, when a series of attacks have occurred, surgical procedure is indicated, as in no other way can the *chronic ulcer be cured.* The type of operation depends on the conditions found, and the methods are as follows:

1. Excision of the ulcer. Simple excision. Finney's operation. Excision should be practised when the ulcer is small and the gut is not narrowed by the operation.

2. Gastro-enterostomy. *Infolding the ulcer, so as to permanently narrow the lumen of the gut* and prevent the exit of food through the pylorus, should be *carried out at the same time.* If obstruction is

already present from contraction of the ulcer, then infolding is unnecessary.

3. Resection of the duodenum with or without the pyloric portion of the stomach.

4. Resection and end-to-end anastomosis, the pylorus being left intact. Posterior *gastrojejunostomy is the best operation.* Anterior gastro-enterostomy, Roux's operation, or Moynihan's modification may be required.

*Stenosis with Motor Disturbances and Dilatation of the Stomach.*—Stenosis of the duodenum near the pylorus or at the pylorus, from contraction and connective-tissue formation from an ulcer, produces dilatation of the stomach with marked disturbance in motility, vomiting, peristaltic unrest, etc. All cases of organic stricture are surgical and gastro-enterostomy is indicated under such conditions.

Acute and subacute perforation require immediate operation. With *chronic perforation* (periduodenal abscess), whether localized or whether it has burrowed, the abscess should be freely opened. These patients often remain with a duodenal fistula and die from inanition, unless further surgical procedure is undertaken. Gastro-enterostomy in such event is indicated and with it an infolding at the pylorus or of the stomach in front of the pylorus, so that food is thus compelled to pass through the new opening and can no longer escape through the duodenal fistula.

Adhesions and obstructive jaundice require operation.

If the patient have recurrent large hemorrhages and be apparently bleeding to death, operation is at once indicated.

## INTESTINAL ULCERS FROM CUTANEOUS BURNS

Ulcers from extensive burns (cutaneous) generally occur in the upper transverse duodenum, seldom lower down. Rarely an ulcer may occur in the stomach or other part of the intestines. There may be a single ulcer or five or six of them. There is considerable loss of tissue in some cases, in others erosions, and at times inflammation of the mucous membrane. The shape of the ulcer is irregular and dentate or long and narrow.

The course is very acute, the result generally fatal; hemorrhage or perforation occurring within one to two weeks after the burn or even within two to three days. The condition is probably due to septic embolism. Operate if there is perforation. In *mild cases* the treatment is that of duodenal ulcer. Intestinal hemorrhage and local tenderness are the salient symptoms.

## EMBOLIC AND THROMBOTIC ULCERS

Parenski[1] first described this condition. These ulcers originate from emboli, which are carried into the small branches of the mesenteric arteries from an endocarditis or atheromatous degeneration of the aorta, from an abscess focus or foci, or from thrombosis, as a result of endarteritis. They occur in the jejunum, ileum, colon, and also in the duodenum.

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[1] Wiener med. Jahrbücher, 1876, Heft. 3.

If the embolus is aseptic, infarction with hemorrhagic infiltration occurs and necrosis results, with the production of an ulcer. The ulcers, as a rule, are small, circular, or irregular in outline. Occasionally the whole thickness of the intestines may become involved, so that peritonitis of a fibrinous or purulent type occurs, or at times perforation. These ulcers occur in the area of distribution of the occluded vessels.

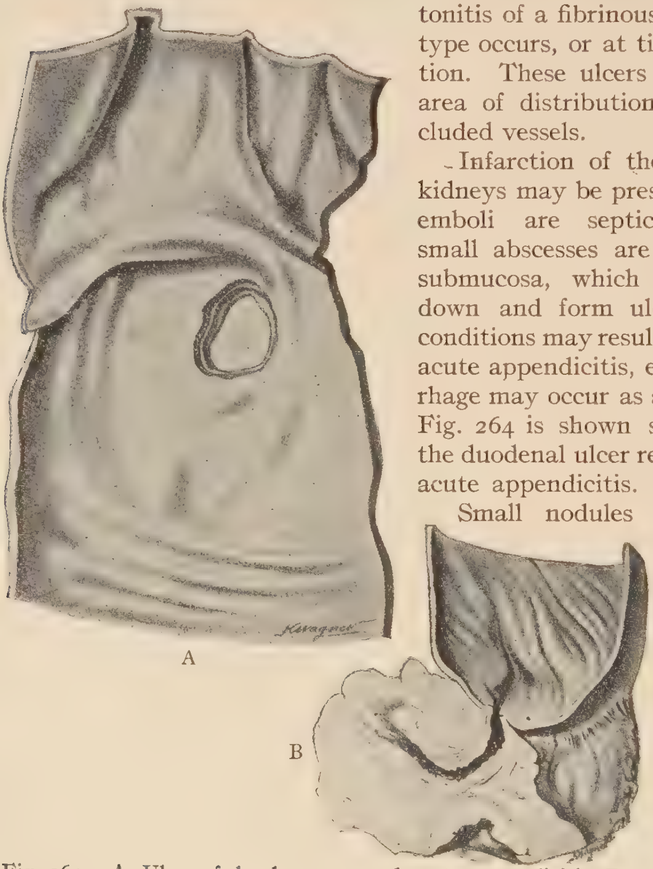

Fig. 264.—A, Ulcer of duodenum secondary to appendicitis; B, primary diseased appendix.

Infarction of the spleen and kidneys may be present. If the emboli are septic, numerous small abscesses are seen in the submucosa, which may break down and form ulcers. These conditions may result from sepsis, acute appendicitis, etc. Hemorrhage may occur as a result. In Fig. 264 is shown such a case, the duodenal ulcer resulting from acute appendicitis.

Small nodules (miliary abscesses), consisting of round cells surrounding a blood-vessel, are at times found postmortem in fatal cases, not yet having broken down into ulcers. Colicky pains, tenderness, diarrhea with blood and pus, occurring in cases in whom embolic processes can be discovered in other organs, or when a cause for emboli can be found, render the diagnosis of embolic ulcer probable.

Intestinal ulceration occurring with multiple degenerative neuritis probably belongs to the class of thrombotic ulcers, there being degenerative changes (arteritis) influenced by the neuritis (trophic). Fracture of the spine has resulted in intestinal ulceration.

## AMYLOID ULCERS

This type of intestinal ulcer is rare, and would only be suspected when associated with amyloid degeneration of other organs, such as of the liver, spleen, in cases suffering from long-continued suppuration, cachexia, tuberculosis, syphilis, rickets, or leukemia. There would be diarrhea, symptoms of ulcer, and deficient absorption from the intestines. Amyloid ulcers may be found in any part of the intestinal tract, though more usually in the small intestine. They are generally multiple and may involve large areas. They are from the size of a pea to large girdle ulcers, nearly circumscribing the bowel.

Leube believes the ulcers originate from circulatory disturbances due to amyloid degeneration of the walls of the small arteries. The vessels of the mucous membrane are first affected, but the process may involve the entire coat of the bowel. The ulcers have no tendency to heal. Other areas of intestines between the ulcers may be in a condition of amyloid degeneration. The mucous membrane is pale and waxy in appearance and some of the villi are missing.

On post mortem a weak solution of iodin gives a mahogany-brown color, the test for amyloid degeneration. The addition of sulphuric acid turns it violet or blue.

## TUBERCULAR ULCERS AND INTESTINAL TUBERCULOSIS

Tuberculosis is a quite frequent cause of ulceration of the intestines. The infection may be: (1) Primary in the intestinal mucous membrane; (2) most commonly secondary to diseases of the lungs; and, rarely, (3) secondary to tubercular peritonitis.

*Primary intestinal tuberculosis* occurs most frequently in children, and with it may be associated tuberculosis of the mesenteric glands or tubercular peritonitis.

R. Koch believes that bovine tuberculosis differs from human tuberculosis, and that infection from diseased milk or milk of tuberculous cattle hardly ever occurs, and it is unnecessary to take any precautions. Von Behring takes the opposite view.

In the Charité Hospital in Berlin there were only 10 cases in ten years. In 3104 cases of tuberculosis in children, there were 16 of primary infection. There have been investigations recently reported which are suggestive that infection through the intestines is more common than we suppose; notably, Macfayden found tubercle bacilli in the mesenteric glands of 5 out of 20 children post mortem, with no tubercular lesions elsewhere; and Ravenal in 8 cases out of 25.

Recent consensus of opinion holds that primary intestinal infection occurs through tuberculous milk. Milk from an infected nurse or mother may also be a cause. The meat of tuberculous cattle probably plays a lesser rôle, as it is generally thoroughly cooked. There is no reason why other raw food products may not be occasionally a source of infection, especially if exposed to infection by flies or sources of contamination. In adults primary intestinal tuberculosis is rare (in the Munich Pathologic Institute 1 in 1000 cases). The lower ileum is usually first involved and then the rest of the small intestine and colon.

**Symptoms.**—The condition may begin with irregular diarrhea, slight fever, and colicky pains. Rarely, hemorrhage may be the first symptom. At first the symptoms may simulate a chronic catarrh. Until subsequent emaciation becomes marked or an involvement of the lungs occurs, the condition may not be suspected. The stools in every doubtful case should be examined for Koch's tubercle bacillus, which is diagnostic. The tuberculin test, by injection or by the ocular method, should be made.

A deceptive condition is when the ulceration begins in the cecum, and there are symptoms suggestive of appendicitis, with tenderness in the right iliac fossa, constipation, or irregular diarrhea. Osler reports, in his primary cases of intestinal tuberculosis, occasional fatal hemorrhage or perforation, with the formation of pericecal abscess or perforative peritonitis, or, rarely, partial healing, with great thickening of the intestinal walls and narrowing of the canal (chronic hyperplastic tuberculosis).

## SECONDARY TUBERCULAR ULCERS OF THE INTESTINES (TUBERCULOSIS)

These are very common in chronic pulmonary tuberculosis.

Frerichs[1] found tuberculosis of the ileum in 80 per cent. of these cases. In 566 of 1000 Munich autopsies secondary tuberculosis was found in the ileum, cecum, or colon, and in all but 3 the lungs were involved. Swallowing tuberculous sputum is the cause.

The lowest part of the ileum is the chief point of infection with ulcers. It often extends to the cecum, colon, or rectum, and upward to the jejunum or even duodenum. Tuberculous ulceration has even occurred in the stomach. At times tuberculous ulcers develop primarily in the colon. The development of the tuberculous ulcer is preceded by the formation of a miliary tubercle. It usually begins in the solitary or agminated follicles. Caseation and necrosis of the tubercle lead to ulceration. In Peyer's patches only isolated follicles are at first involved, while in typhoid and intestinal catarrh they are uniformly affected. Ulceration at first occurs in certain points of the plaque, but later it becomes entirely involved, and the ulcer, therefore, may be ovoid. This occurs in the ileum. In the jejunum and colon they may be round, but usually lie transverse to the long axis. The chief characteristics of the tuberculous ulcer are as follows:

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[1] Beitrage zur Lehre von der Tuberculose, Marburg, 1882.

It is irregular in shape, more rarely ovoid, and generally extends along the transverse axis (girdle ulcer); the margin is a light red color; the edges and base are infiltrated and often caseous. The submucous and muscular coat are usually involved, and on the serosa are miliary tubercles or a marked tubercular lymphangitis. The serosa is reddened and thickened, covered with layers of fibrin, and is often adherent to the mesentery or other loops of the intestines. As compared with the great frequency and number of tuberculous ulcers, perforation is, therefore, not frequent. There may be stenosis from cicatrization of the ulcers and this may be multiple. They do not, as a rule, show much tendency to heal. Swelling and tuberculous infection of the mesenteric glands are often present.

Secondary infection of the intestines from the peritoneum may produce tuberculous ulceration. The affection may be primarily in the peritoneum, or extend from the Fallopian tubes or mesenteric glands. The intestinal coils may mat together, containing caseous, suppurating foci.

There may be the peculiar localized form of tuberculous tumor from a chronic hyperplastic tuberculosis occurring in the ileocecal region, to which I have already referred. It may simulate a new growth and cause constriction of the lumen of the bowel.

There may be a chronic hyperplastic tuberculosis of the intestines with thickening of the gut. There is no definite tumor to be felt, but the induration in the right iliac fossa, when it occurs there, is similar to a recurring appendicitis. It may attack other parts of the intestines. Tuberculosis of the rectum may occur, with *fistula in ano.*

Tubercle bacilli in the stools are diagnostic of these conditions when found; otherwise they are difficult of diagnosis unless by operation. The symptoms and treatment of tuberculous ulcer (tuberculosis) of the intestines will be described at the end of this chapter.

### CATARRHAL AND FOLLICULAR ULCERS

These types of ulcer occur in the course of catarrhal inflammation of the intestinal mucous membrane, and are described in that chapter.

### ULCERATIVE COLITIS

This special form of ulceration of the colon was first described by Hale White, colitis occurring especially in institutions and insane asylums. Probably the greater percentage of these so-called cases of colitis, such as occur in institutions, are true dysentery.

Vedder and Duval,[1] while working under Flexner, found that institutional outbreaks were due to Bacillus dysenteriæ. Osler and J. P. Tuttle differ in this regard and classify it as non-dysenteric. The symptoms and treatment are of dysentery.

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[1] Journal Exp. Med., Feb. 5, 1902, vol. vi.

### STERCORAL OR DECUBITAL ULCERS

These ulcers are produced by the pressure of hardened and stagnating feces on the mucous membrane, resulting in necrosis and purulent inflammation. They develop particularly in the cecum, flexure of the colon (hepatic and splenic), sigmoid flexure, and the rectum, where stasis is most apt to occur. Fecal concretion in the appendix is fairly common and may produce ulcer and appendicitis. They sometimes develop in chronic intestinal stenosis above the seat of stricture.

### ULCERS IN ACUTE INFECTIOUS DISEASES

Under this group are the specific ulcers of typhoid and dysentery; diphtheritic ulceration; ulcers of sepsis; rarely duodenal ulceration in erysipelas; in varioloid; ulcer of the duodenum in pneumonia, a rare occurrence. In acute pemphigus and in pellagra isolated cases have been reported, also in purpura.

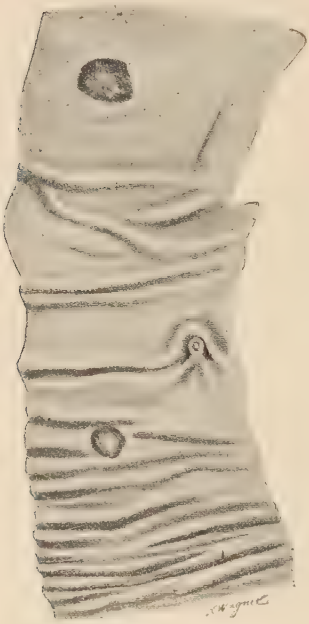

Fig. 265.—Uremic ulceration of the duodenum (Gaultier).

### ULCERS IN CONSTITUTIONAL DISEASES

In acute leukemia, lymphatic tumors may occur in the intestines, especially in the ileum, and break down, with the production of ulcers. This is rare in the chronic type. Intestinal ulceration has occurred in scurvy and a few isolated cases are reported in gout.

### TOXIC ULCERS

Intestinal ulcers occur in nephritis, with uremic symptoms associated with the intestinal catarrh. They lie chiefly in the large intestine. They may occur in the duodenum (Fig. 265).

In poisoning with mercury they are also found, even when it is not administered by mouth or rectum, but by inunction.

Changes in the blood have been held responsible for these conditions. With nephritis the intestinal catarrh is probably a factor. It is believed by many that the mercury reaches the intestines by excretion in the bile, and produces direct inflammation with resultant ulcer, in which decomposition or intestinal bacteria play a part, since we find in some cases a pseudodiphtheritic membrane.

## SYPHILITIC, GONORRHEAL, AND CANCEROUS ULCERS

Syphilitic ulceration of the intestines is rather rare. In the small intestine it may be found in a young infant with inherited syphilis. Ulcers originate in the lymphatics or in the mucosa or submucosa, from a gumma which gradually breaks down. A few cases of syphilitic ulcer of the small intestine in an adult have been reported by Klebs, Osler, and Birch-Hirschfeld. These ulcers are rarely encountered in the large intestine, except in the rectum in its lowest part. Primary chancre of the rectum has been observed. If feasible, the test for the Wassermann reaction should be made to confirm the diagnosis. The physical examination and history of the case are important.

Condylomata and gummata may break down, and by cicatrization give rise to stricture of the rectum.

Virchow notes that syphilitic ulcers are of slight depth and have a smooth base. They are more frequent among women than men.

Polchen[1] found 190 cases of stricture among women out of 219 cases, but believes that many of these result from ulcerations caused by gonorrheal infection, from abscess of the Bartholin glands, or from fecal pressure (decubital ulcer), or local traumatism from the syringe-tip.

Ulcers from gonorrhea, traumatism, and hemorrhoids occur in the rectum and are referred to under Proctitis. Ulceration of intestinal carcinoma may occur, or metastatic nodules may break down and ulcerate.

## INTESTINAL MYIASIS

A case of fatal ulceration of the colon due to maggots has been reported.[2]

## GENERAL SYMPTOMS OF INTESTINAL ULCERATION

Our readers must take into consideration the special types of ulcer, their etiology, and the history of the case in making the diagnosis.

In some cases this is quite difficult, as the symptoms are not constant. The salient diagnostic points of intestinal ulcer are as follows:

1. The appearance of pus in the stool. This is frequently in small quantity and in the form of minute grayish-white lumps, which appear under the microscope as closely aggregated masses of pus-cells, and are especially important. At times it is only microscopic.

2. Necrotic pieces of the intestinal mucosa, such as shreds of tissue from the intestinal wall, which must be differentiated from membranous or shred-like particles of food, occur in some cases.

3. The appearance more or less frequently of blood in the stool,

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[1] Virchow's Archiv., Bd. 127.

[2] Schlessinger, Wiener klin. Wochens., January 9, 1901.

unchanged and in large amount as pure blood, or changed, coffee-ground, or *occult blood.* In some cases blood is only shown under the microscope, or by hematin crystals, or by Weber's or the benzidin test. Gastric ulcer and vicarious hemorrhage must be excluded.

4. The constant appearance of tubercle bacilli in the stools in connection with diarrhea and increasing emaciation are diagnostic of tuberculous ulceration; also the tuberculin reaction.

5. Persistent diarrhea (with local tenderness) and pain of a greater or lesser degree over a definite region of the abdomen, extending over a considerable period of time, associated with the presence of pus and blood in the dejecta.

6. Tenesmus, with pus and blood in the stool, suggests ulcer, probably in the rectum, and visual examination makes the diagnosis.

*The presence of mucus is not diagnostic of ulcer.*

**Diarrhea.**—This is present in a large number of cases. It is dependent on the site of the lesions; ulcerations of the small intestine, cecum, or ascending colon probably do not produce diarrhea unless there is a complicating catarrh, amyloid degeneration, or some special infection like typhoid fever. Even in the latter we have constipated cases. Ulcers of the lower colon and rectum usually produce diarrhea, but even here it may occasionally be absent.

**Blood.**—Pure blood may be passed in large amounts in simple duodenal ulcer, in ulceration from burns, and in typhoid. Large hemorrhages at times occur from dysenteric ulcers. The hemorrhages from catarrhal and tuberculous ulcers are, as a rule, not as large.

The blood may be bright red or dark, or of coffee-ground color, or may be only determined microscopically as blood-corpuscles or hematin crystals, or by tests for occult blood.

Intestinal ulcer may be present without hemorrhage, and other conditions may produce intestinal hemorrhage, such as liver cirrhosis, etc. Repeated examinations, however, will generally show occult blood.

**Pus** is diagnostic of intestinal ulcer. It is also found in connection with ulceration accompanying neoplasms of the intestines, and in abscesses which open into the intestines. It is generally in small amounts. Occasionally no pus is found, as in the case of duodenal ulcer; or the ulcer may be single and high up, so that the pus will disappear. If small grayish-white specks are discovered in the stool, and under microscopic examination they are found to be pus, the diagnosis of ulcer is established. Pus is often only found by the microscope.

**Mucus** found mixed with the intestinal contents is the result of *associated intestinal catarrh.* We can draw some conclusion from the relative amount of pus, blood, and mucus. *Mucus is never diagnostic of ulcer.*

Pure pus is also found in a diphtheritic process of the bowel or from perforating abscess.

Blood, pus, and mucus occur in dysentery and in carcinoma of the lower colon or rectum.

**Shreds of Tissue.**—They consist of mucous membrane and are differentiated from particles derived from food; they occur most frequently in dysenteric ulcer and not, as a rule, in the slower type, as in tuberculosis; or in the more rapid, as in typhoid.

**Tubercle Bacilli.**—Generally are diagnostic of tuberculous ulcer, in connection with the other symptoms. Rarely sputum may be swallowed and pass through without infection. Absence of bacilli does not always prove absence of tuberculous process. The injection of tuberculin or the ocular test (conjunctival) aid diagnosis in the doubtful cases.

**Pain.**—This is occasionally absent. If pain exists in a circumscribed spot for a long time and is increased on pressure, it is probably due to ulcer. It may at times be caused by local peritonitis.

Ulcers of the rectum produce tenesmus, which is quite characteristic. *Rectal examination should always be made.*

**Fever** is present in the tuberculous type, dysentery, etc., but not in all types of ulcer, being dependent on the etiologic cause.

**General Nutrition.**—This may not be disturbed by a few small ulcers, but marked ulceration, especially of the tuberculous type, leads to great emaciation, as the intestinal contents are rapidly propelled and also normal absorption is interfered with.

**Perforation** with general peritonitis, local peritonitis, or encapsulated abscess may occur. Stricture may result from ulcers; also pancreatitis.

**Prognosis.**—The prognosis depends on the etiology.

## TREATMENT

Hemorrhage should be treated as when from duodenal ulcer. The primary cause should receive appropriate treatment; in uremic ulcers, the nephritis; in syphilitic ulcers, by hypodermics of bichlorid of mercury; mercurial inunction; protiodid, bichlorid, and iodid of potassium, or "606."

Heat or cold can be applied for the pain and rest in bed for the severe cases.

In tuberculous ulceration, out-of-door life and change of climate are important. Beechwood creosote, 2 minims (0.118) t. i. d., guaiacol carbonate, carbonate of creosote, and creosal (tannosal) are useful. Average doses of these remedies are 10 grains (0.6) t. i. d.

Diet is important; it should be non-irritating and chiefly liquid, such as milk, koumiss, matzoon, bacillac, kefir, fermillac, lactone-buttermilk, raw eggs beaten in milk; soft-boiled eggs, broths, chicken soup, mushes, etc. Fats, such as butter, emulsion of mixed fats (Russell's), cream, etc., are of value.

Some cases can take sweetbread, scraped beef, calves' brains, cocoa, tea or weak coffee, milk toast, etc.

Sanatogen (flavored) and malt-tropon can be given in broths or soups.

Compound tincture of catechu, chalk mixture, opium, etc., are useful for severe diarrhea. The following are excellent:

| | | |
|---|---|---|
| ℞. | Camph. tinct. opii<br>Bismuth. subnit. | āā ℥ss (16.0); |
| | Mist. cretæ | ℥ij (60.0); |
| | Aq. destil. | q. s. ℥iv (125.0).—M. |

Sig.—Shake. Dose, ʒij (8.0), every three hours.

| | | |
|---|---|---|
| ℞. | Tr. opii | mx (0.59); |
| | Mist. cretæ | ʒj (4.0); |
| | Comp. tinct. catechu | q. s. ʒij (8.0).—M. |

Sig.—Dose, ʒij (8.0). Administer every three hours.

Bismuth subnitrate, 15 to 30 grains (1.0–2.0), three or four times a day, or bismuth subcarbonate or bismuth salicylate, 10 to 15 grains (0.6–1.0), may be substituted. *Avoid opiates as much as possible.*

Tannigen, bismuth subgallate, tannalbin, and tannocol are useful in average doses of 10 to 15 grains (0.6–1.3) three or four times a day.

High injections of silver nitrate, 1 : 3000; thymol, 1 : 2000; salicylic acid, 1 : 300; boric acid, 1 : 500; tannic acid, 1 : 1000; and protargol, 1 : 1000, are of service in rectal and colonic ulcers.

Carbolic acid *should not be used,* and bichlorid of mercury (1 : 10,000) by recurrent irrigation only in typhoid in the diphtheritic form of colitis. Special local treatment is necessary for proctitis, as already described.

## DISEASES OF THE BLOOD-VESSELS; EMBOLISM AND THROMBOSIS OF THE MESENTERIC ARTERIES AND VEINS (INFARCTION OF THE BOWEL)

Embolism and thrombosis of the mesenteric arteries are comparatively rare affections.

When the mesenteric vessels are blocked by emboli or thrombi, infarction follows in the territory supplied by the vessel, which may continue on to gangrene or perforation and peritonitis. If only a few small vessels are occluded there may be few if any symptoms, and the circulation may be re-established. Welch[1] states that about 70 cases of embolism or thrombosis of the mesenteric arteries have been published, while Gallavardin[2] has collected 83 cases, of which 63 were of embolism.

As the clinical symptoms of obstruction of the mesenteric arteries and veins can scarcely be differentiated during life, the symptoms being much the same, and since careful study of their etiology may aid in diagnosis, it is advisable to describe them together. Including both arterial and venous mesenteric obstruction, Jackson, Porter, and Quimby have studied about 30 cases in Boston, and have collected 214 cases in all.

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[1] Allbutt's System of Medicine, 1894, vol. ii; also vol. iv.

[2] Gaz. des Hôp., Paris, 1901, p. 929.

**Etiology.**—*Embolism and Thrombosis of the Mesenteric Arteries.*—In a majority of cases occlusion is due to embolism and rarely to thrombosis.

The chief source of the emboli are traceable to endocarditis (valvular lesions) or atheroma of the aorta, or rarely from a pulmonary thrombosis or aneurism of the aorta.

*Thrombosis.*—Local changes in the vessels, as endarteritis (described by Litten), syphilitic endarteritis, injury and pressure from calcareous glands, have been given as causes. Rolleston refers to periarteritis nodosa, a rare condition in man, with the production of multiple aneurysms, which produces pain, colic, and diarrhea with ulceration. The superior mesenteric artery is more often affected. Verminous aneurysms cause infarctions in the horse.

**Etiology of Thrombosis of the Mesenteric Veins or the Portal Vein.**—Welch[1] has collected 32 cases, and has demonstrated that the superior mesenteric vein is more often affected than the inferior mesenteric. Among the causes are pressure on the portal vein, as in cirrhosis or cancer of the liver; neoplasms of the abdomen; chronic peritonitis, with formation of constricting tissue; local pressure or incarceration of the intestines; suppurative inflammation of the portal system as a result of infection, as in appendicitis, intestinal ulcer, or dysentery. Traumatism may produce inflammation and thrombosis. Mayland[2] shows that it may occur after operation; from tuberculous peritonitis, or diseased mesenteric glands. Hemorrhagic infarction of the intestines, as a rule, occurs with venous thrombosis, and the general symptoms are the same as due to arterial occlusion; hence, the comparative study of the etiology of these different conditions is important for diagnosis.

We would note that in a few rare instances Welch and Rolleston have reported exceptional conditions; the former, that acute portal obstruction has caused hemorrhagic infarction of the intestines, without thrombosis of the mesenteric vein; and the latter, thrombosis of both veins without infarction.[3]

Elliot, Sprengel, and Berg have described cases, and H. Fisher[4] reports two cases that are of interest.

**Pathology of Occlusion of the Superior Mesenteric Artery.**—The changes resulting from occlusion of this vessel or its branches are hemorrhagic infarcts and peritonitis. If only small branches are occluded, the results are embolic or thrombotic *ulcers of the intestines*, to which article I refer my readers.

The transverse duodenum, ileum, jejunum, caput coli, ascending and transverse colon are supplied by this vessel, and lesions of the

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[1] Allbutt's System of Med., vol. v.

[2] Brit. Med. Jour., 1901, vol. ii.

[3] Rolleston, Trans. Path. Soc., vol. xlii.

[4] Archiv. of Diag., N. Y., Oct., 1909.

intestines occur in that portion of the bowel supplied by the occluded branch or branches. It usually involves a continuous segment of the gut, quite frequently in the lower jejunum and ileum; but if smaller vessels or branches are occluded, there may be lesions scattered in several places over the bowel, with healthy segments between them.

The superior mesenteric artery has such long and small branches that it acts functionally, like a terminal artery, so sufficient blood cannot be supplied to the intestines if it or one of its branches be occluded.

Welch and Mall[1] have demonstrated experimentally that if a branch be occluded, the blood which produces the hemorrhagic infarction is derived from anastomosing arteries and not from regurgitation from the portal vessels, as was formerly supposed.

The following are the clinical appearances in the intestines: Arteries empty, except at obstruction. There are venous hyperemia; edema and necrosis; hemorrhages in the mucous membrane and mesentery. The mucosa is a dark red and finally becomes necrotic, of a brownish-green appearance. The intestinal canal contains extravasated blood, either fresh or tarry looking. Necrosis of the intestines is present.

The serous coat is inflamed, not only in the affected area but also over the healthy intestines, and the coils may become adherent and be covered with fibrin. There may be a blood-stained or purulent exudate in the peritoneal cavity. Occasionally gas may be present in the cavity, due to the Bacillus aërogenes capsulatus or to emphysema of the mucosa.

**Clinical Symptoms.**—Kussmaul and Gerhardt[2] first clearly described these some years ago.

There are two types, the most frequent characterized by *hemorrhage from the intestines*; the second, *simulating intestinal obstruction*, with or without peritonitis.

Usually the onset is sudden, frequently with violent, colicky pains in the region of the umbilicus, which may gradually become diffuse, and, at the same time, there is tenderness on pressure over the abdomen. We must remember that in some cases there is an absence of pain.

Vomiting may accompany the pain. In some cases there may be blood in the vomitus. Diarrhea may begin shortly after the pain and become extreme—mucous, watery, and at last bloody—or an intestinal hemorrhage may occur at the commencement, with dark-brown or tarry stools, which at times may have a fetid odor. The blood may not always be voided, but retained, and the patient may have the symptoms of hemorrhage—collapse, cold extremities, rapid and feeble heart, and subnormal temperature.

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[1] Johns Hopkins Hosp. Reports, vol. i.

[2] Würzburger med. Zeitschr., 1863 and 1864, Bd. iv. and v.

In the other type of cases the patient may have signs of acute intestinal obstruction; a history of constipation for some days; distended and painful abdomen; vomiting, at times feculent; severe abdominal pains; collapse and peritonitis. The attack is clearly due to intestinal paralysis.

Sprengel groups the cases according to their pathological causes: First, hemorrhagic infarct; second, anemic gangrene. Under *the first group one* would expect: Bloody ejections per rectum or os; serohemorrhagic exudate in the peritoneal cavity; thickening of the intestinal wall, due in part to hemorrhage; gradual paralysis of the intestine; late peritonitis, local or diffuse. Under the second group (anemic gangrene): Early paralysis of the intestinal walls from local necrosis, early obstruction (early distention of abdomen, following distention of afferent gut); early peritonitis, diffuse or local, depending also on the occurrence of early or late perforation; inflammatory exudate in the peritoneal cavity; all these symptoms can also be met with in acute invagination and in other forms of acute obstruction.

**Diagnosis.**—The following may be of service: An intestinal hemorrhage from no apparent cause: colicky pains of great severity, tympanites and tenderness; evidences of effusion into the peritoneal cavity; the discovery of simultaneous embolism in other vessels or of an endocarditis. In many cases, however, the salient symptoms are not all present; and in the type simulating obstruction the true cause cannot be determined.

**Prognosis.**—This is generally fatal in occlusion of the superior mesenteric artery. In rare instances recovery may be possible.

Treatment will be described at the end of the chapter.

**Embolism and Thrombosis of the Inferior Mesenteric Artery.**—In 83 cases of Gerhardt, in only 5 instances was the inferior mesenteric artery obstructed; in 4 cases by emboli and in 1 by thrombosis; and in 2 of these the superior mesenteric artery was also obstructed. *This condition is rare.*

*Symptoms.*—The chief symptoms are pain, tenesmus, and bloody stools. Gerhardt states that the blood is bright red and that in obstruction of the superior vessel it is darker, but I believe it generally dark. The descending colon, sigmoid, and rectum, or parts of these are affected, the mucosa becoming red, succulent, and containing effusions of blood. The mucous membrane may be loose and detached in places and hemorrhagic. There are not the serious lesions, as a rule, as in occlusion of the superior mesenteric artery. The artery (inferior mesenteric) is not an end artery functionally, and the circulation is generally re-established. Rarely there may be infarction or gangrene with perforation.

**Thrombosis of the Mesenteric Veins or Portal Vein.**—This condition is extremely serious.

*Anatomic Findings.*—The arteries are distended (thus differing from the superior mesenteric artery obstruction); thrombotic processes are found in the mesenteric vein or its branches or in the portal vein. The intestines show similar conditions to obstruction of the arteries—infarction, necrosis, hemorrhage, peritonitis, etc.

*Symptoms.*—The clinical picture may present the same type as in obstruction of the mesenteric arteries—colicky pains, tenderness, blood, diarrheal movements, etc. Rarely there may be hematemesis, and occasionally constipation. The etiology, such as appendicitis, diseases of the liver causing pressure on the portal vein, abdominal neoplasms or constricting bands, producing pressure, etc., and the exclusion of the causes of embolism, may aid our diagnosis.

*Treatment.*—Temporarily employ an ice-bag to the abdomen; stimulants, morphin, $\frac{1}{4}$ grain (0.016), and ernutin, 5 minims (0.296), by hypodermic; gelatin solution, calcium chlorid, or lactate of calcium to check hemorrhage, as described in Dysentery.

Immediate resort to laparotomy and resection of the diseased area is indicated, if possible. Cases of recovery after this procedure have been reported. Elliott resected 48 inches of intestines in one case with recovery. Protoclysis is of value for the sepsis.

## CHAPTER XXVIII

# NEOPLASMS OF THE INTESTINES

## MALIGNANT GROWTHS

(*Synonym.*—Neoplasmata Maligna Intestini.)

THOUGH many have claimed that intestinal cancer is a comparatively rare disease, recent statistics show that it has increased in frequency and is more common than has been generally supposed.

Among the malignant tumors met with in the intestines, carcinoma is by far the commonest. Sarcoma and lymphosarcoma are also found, and will be described in a separate section.

## CARCINOMA OF THE INTESTINES

**Etiology.**—The cause of intestinal cancer, like that of cancer of other organs, is still unknown. There have been many theories advanced, such as the embryonal origin, parasitic infection, the theory of irritation, etc. The traumatic theory (irritation as the cause), in so far that it might *hasten the development* of the condition, is afforded a certain amount of plausible support, in view of the fact that the disease occurs most frequently in those parts of the bowels in which the feces are retarded, and hence act as a source of irritation. The points of selection for the development of cancer are the same as those in which the so-called decubital or stercoral ulcers most frequently develop, and we may assume they probably originate from the cicatricial tissue of these ulcers, in some cases at least, or on the base of other ulcers.

Billroth has found a carcinoma in the scar of a dysenteric stricture, just as it occurs in the case of carcinoma engrafted on the scar of a stomach ulcer. It will probably eventually be shown to be due to some specific organism in my opinion.

*Sex.*—Carcinoma of the intestines appears to be somewhat more common in men than in women.

*Age.*—In general this lesion is most common between the fortieth and sixty-fifth year.

Contrary to cancer developed in other regions, cancer of the intestines has been encountered quite frequently before the fortieth year. It has been found even in children; several cases being reported at the ages of eleven to seventeen, and 2 cases in children of three years of age.

Maydl, of Vienna, has calculated from records that one-sixth of all cases of intestinal carcinoma occur between thirty and forty years, and one-seventh before the thirtieth year. It is important to remember that *early occurrence is fairly frequent.*

**Situation of the Carcinoma.**—Cancer of the bowel is rare in the small intestine, occurs quite frequently in the colon, and most commonly in the rectum.

From 1882–93, from autopsies in the Vienna General Hospital Riegel states 5 cases were in the duodenum; 6 in the ileum; none in the jejunum; 1 in the vermiform appendix; 14 in the cecum; 63 in the colon; 40 in the sigmoid, and 114 rectal cancers.

At the same hospital, during twelve years, out of 254 cases of cancer of the intestines, Maydl found in *the living* that 224 were cancer of the rectum.

Bryant, in 110 autopsies, found carcinoma of the small intestine 6 times; 7 times in the cecum and ileocecal region; 19 times in the transverse colon, including its flexures; 78 times in the sigmoid flexure and rectum.

Leichtenstern's data show that 80 per cent. of all intestinal cancers occur in the rectum.

Other statistics are given. They all show the preponderance of rectal cancer.

Intestinal cancers are *almost always primary,* and secondary growth by metastasis is extremely rare.

It may occur through direct extension by continuity, as a cancer of the pancreas may extend to the intestines.

Primary carcinomata of the intestines often give rise to metastases in other organs. These are most frequently found in the lymph-glands, especially in the neighborhood of the neoplasm.

Secondary metastases in the liver are quite frequent, no matter where the situation of the carcinoma. The peritoneum, omentum, mesentery, and lungs may be involved, and occasionally the kidneys.

Hauser calls attention to certain peculiarities regarding the metastases in different forms of carcinoma of the large intestine. Colloid carcinoma chiefly involves the serous coat and metastases of internal organs are rare. Medullary tumors involve adjacent lymph-glands, while simple and scirrhous carcinomata, even when small, frequently cause *carcinoma of the liver.*

**Morbid Anatomy.**—Several varieties of carcinoma are found in the intestines, most often the cylindric epithelial-celled carcinoma (adenocarcinoma), starting in the cylindric epithelium of the intestinal glands (follicles of Lieberkühn). It occurs most frequently in the large intestine, as do medullary carcinoma and colloid cancer. More rarely the scirrhous carcinoma is found. The epitheliomatous chancroid occurs in the lower rectum at the anus, and it may involve the perineum and vagina.

In the small intestine the primary proliferation starts from the glands of Brunner; in cases developing from cicatrices, proliferation may start from glandular tubules which have grown deep down into the tissue. The growth varies in consistency, depending upon whether connective tissue or cells predominate; if the former, then the tumor presents a hard consistence (scirrhous); and if the latter, then it is less firm and occasionally soft and succulent. The colloid cancer contains a brown viscid fluid. The scirrhous shows a tendency to ulcerate.

The cancer may form a hard annular induration, as in the colon, or a circumscribed nodule or an ulcerating gangrenous, cauliflower growth, as in the rectum. The nodule may develop into a single large tumor or several smaller masses; the softer tumors usually grow to larger size. At other times the mass may protrude into the intestines, like a polypus; or may infiltrate a large surface of the bowel, so that it becomes stiff and rigid.

In the majority of intestinal carcinomata the surface is ulcerating.

The annular form of the growth is most common, tending to involve the circumference of the bowel. It may develop from a small nodule or, more frequently, on the base of an old annular cicatrix, due to former ulceration.

*Stenosis of the canal is often the result.*

Secondary changes develop; the intestines become dilated through stagnating feces and gas, and the walls hypertrophied above the point of stenosis through overexertion to overcome the obstacle. Catarrhal inflammation and stercoral ulcers develop in the dilated portion of the gut and perforation may ultimately occur.

Below the stricture the intestinal walls are thinner, and if the stenosis is narrow, the intestines may be empty and contracted.

Stenosis of the bowel may be produced by a growth of the cancer into its lumen or by infiltration of the entire wall.

Large masses of very hard scybalæ often accumulate above the seat of obstruction and are difficult to distinguish from the carcinoma proper, so that on autopsy a small growth may be found which *intra vitam* was believed to be of large size.

When stenosis of the bowel occurs, all the symptoms are present which are described under this condition.

Narrowing of the bowel does not always take place. Sometimes the symptoms of stenosis may gradually disappear, being due to ulceration of the neoplasm, so that the canal again becomes patent. As a rule, the tumor tends to grow and fill it up again.

The necrotic process often causes more or less hemorrhage, and in rare cases, if a large vessel is eroded, there may be a fatal issue. The canal may become patent by a direct connection becoming established, through ulceration and adhesions, between two loops of the intestines.

The muscular and serous coats are frequently involved, and peritoneal adhesions develop which may unite the diseased intestines to other portions of the intestines, or to some adjacent organ, which may constitute a serious obstacle to the removal of the growth.

Perforation may rarely occur before the formation of adhesions, with resulting general peritonitis, or there may be a circumscribed abscess formed within the adhesions. Marked displacement of the intestines may be caused by the formation of these adhesions.

A carcinomatous peritonitis may be produced by extension from the serous layer of the intestines, accompanied by hemorrhagic exudation. Perforation into other organs which have become agglutinated to the bowel can occur; thus a fistulous opening forms between the colon and the stomach or bladder, or vagina or uterus, or between the large and small intestines, or from the bowel through the abdominal wall.

The omentum and mesentery may become infiltrated with cancerous masses. A band may be formed by the stiffened omentum, which may cause a knuckling or a twisted bowel. The mesentery may kink and twist the intestines.

**Symptoms.**—The symptoms vary, depending on the position of the neoplasm, the rapidity of its growth, and the character of the cancer. There may be symptoms at first of habitual constipation or of hemorrhoids, or of stenosis of the intestines, or of peritonitis, or icterus, or of cachexia of uncertain origin. It seems preferable to first review the general and then the local symptoms, depending upon the position of the tumor, as occurring with—

1. Carcinoma of the duodenum.
2. Carcinoma of the small intestine. (jejunum and ileum).
3. Carcinoma of the colon (cecum to sigmoid flexure).
4. Carcinoma of the rectum.

**General Symptoms.**—There are symptoms common to all cases of carcinoma, of which anemia and cachexia are the most important. As a rule they occur together, though one may develop before the other. In many patients weakness, pallor, and emaciation are the first signs noticed, and arouse the suspicion of a serious disease. There may be only slight local symptoms, moderate constipation, and a sense of weight or discomfort in the abdomen, with loss of appetite, coated tongue, and slight dyspeptic disturbances. There are loss of weight and a peculiar cachectic appearance. On the other hand, the local symptoms may be the more pronounced, or the local and general symptoms may occur together.

Quite frequently, especially in those cases of carcinoma which run a rapid course, fever is present. I have seen a number of cases in which it is entirely absent. It probably depends upon an ulcerative condition in the growth and absorption of toxic products. The tumor may cause symptoms in adjacent organs by dragging upon them or constricting them. There may be radiating pains from compression of nerves and also disturbances of the circulation, such as edema of the lower extremities.

Symptoms of chronic intestinal obstruction are frequently present, but not in all cases. The symptoms may develop gradually, the constipation increasing, or there may be a sudden stoppage, with all the signs of acute obstruction. The clinical symptoms of cancerous obstruction are similar to those from stenosis of the intestines caused by other processes, and are described in the chapter on that subject.

When acute obstruction suddenly occurs during the course of chronic stenosis a fatal result may ensue within a few days. Fecal retention has been reported in connection with malignant stenosis, lasting even over eighty days,[1] without the presence of fecal vomiting.

Diarrhea is fairly frequent in carcinoma of the intestines. It is of assistance in clearing up the commencing obstruction, and in some cases alternates with constipation.

In some patients the stools appear as small hard balls, cylindric, like a pencil, or flat and tape-like, as if they had passed a strictured point. These are not always characteristic, as they may appear with nervous conditions.

The stools frequently contain pus, blood, and mucus. The appearance of the first two (pus and blood) is significant. If the growth is ulcerating markedly, the stools have a most offensive odor. In this event particles of tumor may rarely be found in the dejecta, which show under the microscope the nature of the growth. They may be of fair size or very small, so that careful examination of the fecal matter may be necessary in order to find them. Irrigation of the bowel is of assistance if the neoplasm is situated in the large intestine.

One of the most important factors in our diagnosis is the detection of the physical signs of a tumor.

The growth is from the size of a walnut to that of a child's head. It is often easily palpable, hard, and usually has an uneven nodular surface.

A marked peculiarity of this type of tumor is its *great mobility* under the palpating hand of the examining physician. Even in the cecum and ascending and descending colon the tumor is distinctly movable as a rule. The mass is usually situated in the lower half of the abdomen, most frequently in the lower left iliac region, as unless three are marked adhesions, the intestines are dragged down there by their own weight. If the growth is in the cecum, the right iliac region is involved.

I have already referred to the fact that on autopsy the tumor is frequently found to be smaller than it was apparent to palpation, this being due to the thickening of the gut above the stenosis and to the fecal accumulation.

Abdominal pain is present, which will be described under Local Symptoms, and the tumor is at times tender.

In some cases simple abdominal palpation is not sufficient, and it may be necessary to examine the patient under an anesthetic.

[1] Cooper-Foster, Med. Times and Gazette, Sept., 1867.

In every case digital examination of the rectum and, if necessary, the passing of a rectal bougie or introduction of a speculum should be employed. In women a vaginal examination should also be made. *I strongly deprecate the insertion* of the entire hand into the rectum under anesthesia, as has been sometimes advised. The *x*-rays are of great value as an aid to diagnosis, the röntgenograph showing the point of stenosis caused by the growth.

When the cancer is fully developed we may have peritonitis, either local or general, as a complication. If the peritonitis is of a cancerous nature, a hemorrhagic exudation and the presence of nodules under the abdominal wall will indicate this fact. Acute perforative peritonitis will be indicated by the usual symptoms of this condition. On the other hand, we may have circumscribed adhesions with the presence of local fecal abscess.

Perforation may occur into adjacent adherent organs which will present special symptoms.

Among the chief communications are:

1. Communication between the colon and stomach. There may be a valvular communication or it may be free. If the direction of the passage is from the stomach into the colon, symptoms of lientery develop—undigested food, such as rice, potatoes, meat, etc., appear rapidly in the stools. Diarrhea occurs shortly after the ingestion of food with evacuation of solid contents. If the valvular action is in the opposite direction, feculent vomiting takes place. If the fistulous opening is perfectly free, then lientery and fecal vomiting occur together or may alternate. This combination of symptoms is pathognomonic. Lavage, especially if the fluid be colored with methylene-blue or carmin, 3 grains (0.194), will demonstrate that the liquid rapidly escapes from the stomach, and is expelled from the bowel without being decolorized or discolored.

If the rectum is inflated with air or $CO_2$ the stomach will be distended with gas, or after injection of 1 to 2 liters (quarts) of colored fluid into the bowel some can be secured from the stomach by aspiration.

2. Communication between the rectum and bladder. Fecal matter and gas escape into the bladder and are voided through the urethra. Severe septic cystitis results, or the urine may escape from the bladder and be discharged through the stools. A carmine solution injected into the rectum or bladder will demonstrate the condition, and will appear respectively in the urine or feces.

3. Communication between the rectum, uterus, and vagina are also met with, and give rise to the passage of fecal matter through these organs, and also to severe inflammation. Injection of weak carmin solution or methylene-blue into the rectum will demonstrate the communication.

4. Communication between the bowel and abdominal wall. There may be a feculent or fetid discharge, or even of particles of

fecal matter, if the communication is with the lower small or large intestines. If the communication is high up, it may be chylous or biliary. This condition appears usually in the last stage of the disease. Injection of carmin red solution through the fistula will appear in the stool. Ulcerative processes (tubercular) may produce similar communication.

The urine is not characteristic, shows indican, occasionally acetone and diacetic acid. Albumin is present. Secondary anemia occurs. Leukocytosis is moderate and eosinophiles are increased.

**Symptoms Due to Position of the Cancer.**—*Cancer of the Duodenum.*—This is a rare condition. There are the anemia and cachexia and pain usually in the right hypochondriac region; this last occurs in the midepigastric region or upper part of abdomen. The tumor, when palpable, is usually found in the right hypochondriac region, near the middle line. If it is in the ascending part of the duodenum, there may be fair mobility; if in the descending or transverse parts, the tumor is only slightly movable.

Nearly all the symptoms are referred to the stomach—anorexia, pains, belching, vomiting, and dilatation of the stomach—and if the mass is in the ascending part of the duodenum, it will hardly be possible to differentiate it from gastric tumor without operation. Blood may be mixed with the vomitus.

Boas makes use of the terms suprapapillary, infrapapillary, and circumpapillary carcinoma, according to the position of the growth relative to the papilla of Vater.

If the carcinoma is suprapapillary, we have stenosis of the upper part of the duodenum, and, as already noted, symptoms identical with pyloric stenosis, from which it can hardly be differentiated.

With infrapapillary carcinoma the gastric symptoms again predominate, but there is stasis of bile and pancreatic juice, and regurgitation of these secretions into the stomach; bilious vomiting is frequent. The vomiting is intermittent and the symptoms those of obstruction. Trypsin should be tested for in the vomit by observing whether fibrin is digested in an alkaline solution. This would *differentiate from a gastrobiliary fistula.*

Circumpapillary carcinoma. In the pure cases surrounding the papilla of Vater, jaundice, anemia, and cachexia slowly increase in severity, without *any gastro-intestinal symptoms*, and pain is usually absent. Some of these cases doubtless have their origin in the wall of the ductus communis choledochus as it passes through the wall of the duodenum as in Fig. 266.

Chills may at times occur and cholangitis may be a complication. Jaundice may vary in intensity or may be intermittent. When the tumor involves the common bile-duct and head of the pancreas jaundice is progressive and continuous. Ulceration may temporarily open a passage for the bile. Gastric symptoms may occur in addition.

The tumor can be palpated deep down in the right hypochondriac region near the middle line. At times it cannot be discovered.

Carcinoma of the duodenum is a disease of late middle or advanced life, occurring most often in males.

The second part of the duodenum is most frequently involved, and next in frequency the first part.

*Carcinoma of the small intestine* (jejunum and ileum) is rare. The symptoms vary, depending on the position of the growth; the higher up, the more marked are the gastric symptoms; the lower down, the more severe the intestinal symptoms.

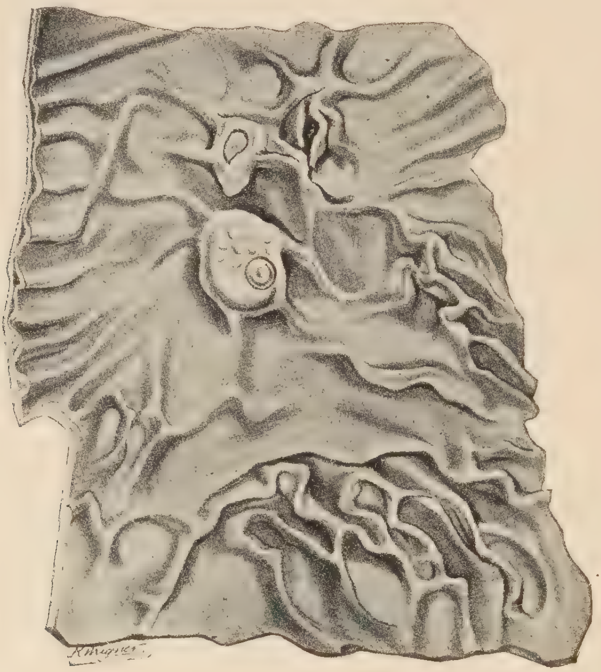

Fig. 266.—Cancer of the region of the papilla of Vater emanating from the common duct (after Letulle).

There may be anorexia and vomiting, or fair appetite and good stomach digestion, but obstinate constipation.

In both conditions there are anemia and cachexia, pain, attacks of colic and constipation, which may alternate with a diarrhea. There may occasionally be hemorrhages from the bowel if the growth is low down. The clinical symptoms of *stenosis* of the intestines may be present. The tumor is at times accessible to palpation, and is, as a rule, extremely *movable*. It may be so much displaced downward that it is difficult to draw an accurate conclusion.

**Local Symptoms of Carcinoma of the Large Intestine.**—Cachexia and anemia are present. Pain is a frequent symptom; it may not be present at first, usually appears later in the disease, and becomes localized; in some cases, running an acute course, no pain appears until the symptoms of acute obstruction begin. Pain is usually localized; it may appear near or at the region of the growth, or occasionally directly opposite, in the abdomen.

The pain may not be severe, but may be more a sense of discomfort. There may be neuralgia of the sciatic or anterior crural nerves. Later pain usually becomes localized, and is increased on pressure, even if the tumor cannot be palpated. It may be due to local peritonitis near the tumor.

*Attacks of colic often occur*, local or diffuse. The pains may be quite severe, are accompanied with constipation, and are relieved by diarrhea or by the passage of flatus. These attacks of colic are frequently caused by the commencing obstruction, and show gradually progressive stenosis. Peristaltic and tetanic movements of the intestines are often associated. Complete obstruction may suddenly develop, or there may be a gradually progressive stenosis with its symptoms.

*Constipation* is one of the marked symptoms; in some cases it may be first, and becomes gradually progressive.

Ten to twenty or even eighty-eight days have passed, according to various observers, before the bowels moved spontaneously or by artificial means. These were, of course, extreme cases. The majority of patients have symptoms of commencing or complete obstruction after coprostasis has lasted a week or ten days. This has been my experience in consultation practice. Recently I saw a case for the first time on the fourth day of coprostasis when acute symptoms were present. Loss of appetite, tension, fulness in the abdomen, and pain accompany the constipation. Spontaneous diarrhea may relieve the condition. If this does not occur, or relief is not afforded artificially, gradual occlusion will take place with its typic symptoms.

In some patients diarrheal movements may occur for several weeks, due to the catarrhal condition of the bowel or ulceration of the growth.

*Stools.*—In some cases of carcinoma the stools may be normal and simply hard in character when constipation is present.

In others they may be in small balls like sheep's dung, flattened, or ribbon shaped.

Mucus shows the presence of *catarrh* of the mucous membrane. *The presence of pus in the stools is of importance*, but only appears when the growth ulcerates; hence its absence does not prove there is no tumor. Microscopic examination for pus is indicated.

*Pus* in some cases may be derived from an abscess cavity opening into the intestines.

The same remarks hold true of blood, the amount found is usually small, and violent hemorrhages are rare.

The appearance of *pus and blood in the stool* in a patient with symptoms of stenosis of the bowel *favors the diagnosis* of malignancy. Occult blood should be tested for if none is visible.

If there is gangrenous disintegration of the tumor, the odor is very characteristic, and occasionally small bits of new growth may be found in the stool.

*Tumor.*—The presence of palpable tumor strengthens the diagnosis. It may be no larger than a nut, or the size of a child's head, and be solid and hard like cartilage. If there is much infiltration, it may give the sensation of a solid and thick cord. These tumors are generally moderately tender on pressure, in some cases quite so; occasionally they are not tender at all. Their great mobility *is characteristic.* On *palpation* they are easily moved about, especially in the sigmoid and transverse colon; fecal accumulation readily displaces the tumor and it may become adherent in an abnormal position. Occasionally peristaltic movements may cause it to appear and disappear during palpation. Respiratory mobility of the tumor also may occur if it be in the transverse colon, or if it be adherent to the liver, spleen, or stomach.

Large amounts of fecal material may accumulate above the growth, and we must clear this material out by irrigation and laxatives, so as to determine the extent of the tumor.

A pure fecal tumor may lead to narrowing and occlusion. These last tumors are more doughy, less firm, and quite frequently multiple.

**Differential Diagnosis.**—We must remember that old, agglutinated masses from recurrent appendicitis may simulate cecal tumor. The history is an aid. Moreover, tumor-like tuberculosis of the cecum may simulate malignant growth. In these cases there may be pulmonary tuberculosis, a previous history of diarrhea, or the presence of tubercle bacilli in the stools. Some cases cannot be differentiated. The tuberculin test aids diagnosis.

Leube called to our attention that chronic inflammation of the sigmoid may occur and be mistaken for carcinoma. Undoubtedly these cases are so-called peridiverticulitis, with chronic thickening, and often stenosis of the sigmoid simulating carcinoma. These patients are generally over sixty years of age, fat and flabby, or they have been fat previous to emaciation. There is often a history of acute left-sided inflammation and generally of prolonged constipation. One must remember the possibility of this condition. *Blood and pus, as a rule, are absent from the stools in these patients.* Often it is impossible to differentiate these conditions, even after resection, except by the microscope.

Cachexia and anemia are always present in cancer.

**Primary Carcinoma of the Appendix.**—This condition has been considered rare, but it has been demonstrated to occur more frequently than was formerly supposed. (See Appendicitis.) Most of the cases give symptoms of relapsing appendicitis or appendicitis; 53 per cent. are under thirty years, and 24 per cent. under twenty.

This condition has only been detected after operation on the appendix. The carcinoma is spheroidal celled.

**Carcinoma of the Rectum.**—This is the most frequent type of cancer, is more readily diagnosed, and more amenable to treatment if early operation is performed. Many of the tumors are within reach of the finger. The symptoms resemble those of carcinoma of the colon, though there are certain signs peculiar to this condition. Rectal examination should always be made.

Pain is more pronounced in rectal carcinoma, both local and radiating to the sacrum, back, bladder, genitals, and *to the sciatic nerve;* there is often a desire to urinate. The pain is usually worse during defecation if the growth is low down, and it may even be agonizing in character, so much so that the patients try to retain the bowel contents as long as possible.

Marked tenesmus is present, and if ulceration, mucus, blood, and pus are evacuated. Constipation is usually present. The higher up in the rectum, the more the symptoms resemble those of cancer of the sigmoid. Tenesmus may be absent if the growth is high up. Diarrhea may at times supervene; occasionally, after sloughing of the tumor, paralysis of the sphincter occurs.

Leube was the first to call attention to the fact that *hemorrhoids frequently developed with carcinoma of the rectum*, and that, too, at an early stage. Piles *occurring suddenly*, and not developing slowly or existing for many years, are suggestive.

Rectal examination is most important, and the finger will usually give the necessary information. One can feel a mass lying directly beneath the mucous membrane or adherent to it. The surface may feel uneven and hard or there may be occasionally a cauliflower or mushroom growth, or a constriction, through which the finger cannot readily pass. If it be barely possible to feel the growth, it is similar in sensation to the cervix uteri.

Blood, pus, or sanious material may be found on the finger if the growth is ulcerating.

Vaginal examination in women must be made to differentiate the source of the tumor. The genito-urinary organs in the male should also be examined.

Simple cicatricial stricture is usually *smooth* and not ulcerated, while a carcinomatous stricture is usually nodular and frequently ulcerated.

It is preferable for accurate diagnosis to excise a portion under cocain for microscopic examination.

If the stricture cannot be reached by the finger, Kelly's speculum is of value. *Never* insert the hand into the rectum.

*Metastatic growths* in other organs are quite frequent from small growths of the rectum; thus, with carcinoma of the liver, a small primary carcinoma of the rectum may be found, even if no symptoms are present.

The bladder and vagina may be involved and fistulous openings occur. Periproctitis (abscess) and fistulæ are rare.

The peritoneum is rarely involved unless the carcinoma is high up.

**Course.**—The termination is death if not operated on. This may occur by occlusion of the intestines and from peritonitis.

It is hardly possible to give a prognosis as to duration. In cancer of the duodenum the general nutrition suffers early, or the case may be very acute and the duration of life is short. In many cases the course varies from six months to two years, while in rectal cancer it may last for three to even four years. Rarely cases come to a standstill and last some years.

In some cases coma (carcinomatosum) appears quite early, probably due to auto-intoxication from intestinal decomposition or from the toxins of cancer. Ewald isolated a body from the urine belonging to the group of diamins in such a case.

Thrombosis may develop and embolus of the lungs occur, with death resulting. Death may occur from exhaustion.

**Diagnosis.**—Presence of a tumor by abdominal palpation or rectal examination, accompanied by cachexia (loss of weight marked) and anemia, with marked constipation and increasing symptoms of stenosis of the bowel; or *cachexia, intestinal disturbances, with no detectable tumor, but with symptoms of progressive stenosis in an elderly person, are suggestive of cancer.* Examination of a tumor fragment, if it can be secured in the stool or from the rectum, is conclusive. Inflation per rectum of the intestines with air or with water is of service. In many cases most of the enema will at once be returned. The *x*-rays are of *particular value* in demonstrating a stenosis due to the growth.

**Prognosis.**—This is fatal, unless a radical operation is performed.

**Treatment.**—Complete and early removal of the growth is indicated. An early diagnosis is important. If abdominal cancer is suspected, *exploratory laparotomy* and complete resection of the growth, with end-to-end or lateral anastomosis, are indicated.

If resection is impossible, entero-enterostomy or enterocolostomy for drainage to relieve symptoms is indicated.

If the tumor is low down in the colon, sigmoid, or rectum and inoperable, then colostomy to relieve the symptoms and prevent irritation of the surface of the cancer.

*In the rectum*, resection, preferably Kraske's operation, is indicated if radical operation is possible.

Palliative curetment and the thermocautery may be employed in some cases. Colostomy under cocain can be performed in the aged and feeble.

Coley's treatment by erysipelas toxins may be tried, but I would not recommend it. It is more successful with sarcoma.

*Diet.*—Soluble foods with little residue, such as milk, broths, bouillon, sanatogen, somatose, cream, butter, rice-gruel, sour milks, matzoon, koumiss, bacillac, raw eggs, etc., are indicated.

Irrigation of the intestines, enemata, and injections with olive oil, and internally, castor oil, magnesium sulphate, rhubarb, cascara, regulin, etc., to keep movements soft; warm applications to the abdomen; morphin, codein, and belladonna are indicated for pain. These last can be given by suppository. Strength should be supported by tonics, iron, arsenic, etc., and pain relieved. Treat complications. Opiates should be employed only when absolutely necessary.

## SARCOMA AND LYMPHOSARCOMA OF THE INTESTINES

Sarcoma of the intestines is rather an infrequent disease, much less frequent than carcinoma. In the course of twelve years, 1882–93, in the Vienna General Hospital, out of 274 autopsies on patients dying of sarcoma, only 3 were sarcoma of the intestines, the ileum, cecum, and rectum. In 61 cases of lymphosarcoma, 9 belonged to the intestines, 1 in the duodenum, 3 in the jejunum, 3 in the ileum, and 2 in the cecum.

Of Libman's cases, 15 were of the duodenum, 18 of the jejunum and ileum, 14 of the ileum, and 3 of the entire small intestine.

Sarcoma occurs as frequently in the small as in the large intestine, and *lymphosarcoma preponderates* in the small bowel,

Of Krueger's 37 cases, 16 were of the small bowel alone and 16 of the rectum; the ileocecal region comes next.

Sarcomata generally attain a large size, even as large as a child's head, and spread over a large part of the intestines.

**Anatomy.**—*Sarcomata usually originate in the submucosa*, the musculature is attacked early, and the serosa is rarely involved. Small round-celled sarcoma is most frequent; occasionally spindle celled. In the rectum they are often melanotic.

Lymphosarcomata start from the lymphatic apparatus, the solitary and agminate lymph-follicles. These occur chiefly in the small intestine.

Sarcomata of the intestines show a rapid progress and metastases are found early. *They do not produce stricture of the bowel*, but a dilatation, and develop in a longitudinal direction along the intestines. The bowel may be enormously dilated. Rectal sarcoma, however, may produce obstruction.

**Age.**—Sarcoma is frequent in young persons; most frequent from twenty to forty years of age; from four to seventy years cases are reported, and one congenital case in an infant three days old.

**Symptoms.**—The clinical symptoms differ from carcinoma. The general health becomes impaired early. The patients emaciate rapidly and become anemic. There is *peculiar want of proportion* between the *rapid impairment of the physical condition* and the *absence of local symptoms*. They become rapidly weak and debilitated (cachexia). There are, as a rule, an absence of abdominal pain and no symptoms of stenosis. Intestinal symptoms are slight—occasionally constipation, alternating with diarrhea.

Exceptionally, stenotic symptoms appear, due to kinking or peritonitic incarceration, or, rarely, ileus. Stenosis occurs in rectal carcinoma.

The tumor appears early, grows rapidly, and can easily be made out.

**Duration.**—The majority of cases die within nine months from general cachexia; only 1 case recorded lived twenty-one months.

**Prognosis.**—This type of tumor proves fatal, and even early operation seems of no value on account of the metastases.

**Treatment.**—Injection of Coley's fluid, the mixed toxins of erysipelas and Bacillus prodigiosus, is indicated, as in inoperable sarcoma in the other regions. One must remember that marked reaction follows the injection in some cases, and some patients cannot undergo the treatment. I have seen the latter occur, and also in another case temporary improvement.

The patient should receive as liberal a diet as possible, and iron, arsenic, and cod-liver oil should be administered. As a rule, no stenosis is present, but if in evidence, operative procedure is indicated. Removal has failed on account of metastases.

## BENIGN TUMORS OF THE INTESTINES (NEOPLASMS)

Benignant neoplasms of the intestines are relatively rare, and their clinical significance is generally slight. They occasionally produce severe symptoms.

These tumors may be attached to the bowel wall by a broad base or by a thin pedicle or stem, and are then termed polypi. The last type is usually of small size, that of a cherry, but rarely as large as a pear, and occur most frequently in the rectum.

The following forms of benign tumor are found: Adenoma, fibroma, lipoma, papilloma, myoma, fibromyoma, angioma, and chylangioma.

**Adenomata** are most frequently met with. They arise from the glands of Lieberkühn and in the duodenum from Brunner's glands; are acinous in structure, and may be attached to the bowel by a broad base or by a pedicle. As a rule they are small, the size of a pea; rarely, the size of a pear. They are red and have a tendency to bleed; usually they are soft and the surface is smooth, though occasionally of a cauliflower appearance. They occur most frequently in the rectum, are usually polypoid, though occasionally annular and flat. They are most common, in this location, in children of from four to seven years, though occasionally found in adults. More than half the cases that occur are in males between sixteen and thirty years of age. Sometimes there is extensive involvement of the intestines, a condition known as polyposis intestinalis adenomatosa; in 1 case several thousand were present. Polypi are never limited to the small intestine. Carcinomatous degeneration of the polypi may occur.

**Fibroma, Lipoma, Papilloma.**—These neoplasms derived from the connective tissues, especially from the submucosa, are very rare. They are, as a rule, lipomatous in structure, and often originate from the appendices epiploicæ. They frequently project into the peritoneal cavity and may twist their pedicle and becomed detached. They may have a broad base or pedicle, are of various shapes, and may grow to the size of an apple. They occur most frequently in the colon and rectum, less frequently in the jejunum, and rarely in the ileum.

**Myoma, Fibromyoma.**—These are very rare, and usually originate from the outer muscular coat of the intestines, chiefly from the longitudinal coat. There are two forms: Spheric nodules, which grow toward the mucous lining and sometimes become pedunculated, the mucous membrane lying loose in front; or they may have a broad thick base, forming a circumscribed thickening, the mucous membrane being adherent.

Rarely the myoma will grow toward the peritoneal cavity.

**Angiomata.**—These are exceedingly rare, and may occur as a telangiectatic mass, or as a flat vascular tumor involving more or less of the bowel wall. Cystic chylangiomata occur in the small intestine.

**Symptoms.**—In many cases there are no symptoms at all, and the condition is discovered accidentally postmortem.

Sometimes they give rise to intestinal hemorrhage. When this occurs in a person in good health, who has never given any history of previous intestinal trouble, the possibility of an intestinal tumor should be thought of. This is especially true in children.

Diarrhea with blood and mucus have been reported in cases of polyposis intestinalis adenomatosa. On account of their small size it is nearly impossible to discover them by palpation.

Symptoms may rarely be produced by narrowing or occlusion of the bowel by the intestinal tumor, or from an intussusception caused by the tumor. When tumors are *situated in the rectum* disturbances are most likely to occur, such as hemorrhage, passage of mucus, tenesmus, and difficult defecation.

Sometimes the mass may be felt by the examining finger, and it may even protrude from the anus and give rise to severe pain. It may occasionally be torn off and passed with the stools, when the symptoms may disappear.

**Course.**—They may remain latent or give symptoms for some years and then be passed per rectum.

**Treatment.**—Intestinal hemorrhage should be treated in the manner already described under Typhoid. The blood will often be bright in color if the hemorrhage is from the colon or rectum; injection of very hot or cold water with 1 dram (4.0) of alum or tannic acid, 30 grains (2.0), locally, or Trémolière's solution, by mouth and rectum; ice-bag to the abdomen; morphin, $\frac{1}{4}$ grain (0.016), and ernutin, 5 minims (0.296), by hypodermic, are indicated.

If the tumors are accessible in the rectum, they should be removed by galvanocautery or by operation.

Ligation and excision, after water infiltration of the pedicle, is an excellent method. When the polypi are higher up, each may be exposed in turn by the aid of the proctoscope. A Gant valve clamp may be snapped on the pedicle of each and they may then be allowed to slough off. This last method is extremely simple and requires no anesthetic. When the growths are very numerous, large or ulcerated, and scattered throughout the colon or sigmoid flexure, cecostomy with irrigation of the bowel, or in some cases extirpation of the diseased portion may be necessary.

## GAS CYSTS OF THE INTESTINES

(*Synonym.*—Pneumatosis Cystoides Intestinorum Hominis.)

Cysts containing air, in the intestines of pigs, were probably first described by Mayer,[1] of Bonn. This condition was also independently described by John Hunter. These intestinal gas cysts have been found quite frequently in apparently otherwise healthy pigs and occasionally in sheep. The theory has been held that bacteria are the cause, or that they are the result of mechanical and physical conditions; for example, that gas may escape through some abrasion into the tissues. Bang[2] first reported this condition in the human being, and Finney the first case in America.

Finney and Welch[3] believe the cyst to be a distinct variety of tumor, the cells of which have the faculty of secreting gas. In practically every case so far the gaseous cyst has been associated with disease of some portion of the intestinal canal, producing an obstruction to the lumen of the bowel. Cysts may be single or multiple and occur in the small and large intestines.

**Pathology.**—There is a dense fibrous tissue framework containing round and spindle cells, and there are clefts and spaces whose walls contain large giant cells with many nuclei. Air spaces are found about these cells, and there is an endothelial lining to these spaces. The blood-supply is rich and hemorrhages may occur in the tissues. Air in the cyst resembles atmospheric air. The tumor is more pronounced in the subserous tissue, though it has been observed in all the layers of the walls.

*Clinically*, there is no definite picture. Crepitation on palpation has been noted. Pain and constipation could be chiefly attributed to other factors.

**Diagnosis** is usually made during operation for some other trouble or at autopsy.

**Treatment.**—If symptoms of obstruction, operation is indicated. After operation for other conditions, they generally diminish in size or disappear.

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[1] Jour. d. prakt. Heilk., 1825. [2] Nord. med. Ark., 1876, viii, No. 18.
[3] Jour. Amer. Med. Assoc., October 17, 1908.

## CHAPTER XXIX

# HEMORRHOIDS; PROLAPSE OF RECTUM; FISSURE

### HEMORRHOIDS

(*Synonyms.*—Phlebectasia Hemorrhoidalis; Piles.)

HEMORRHOIDS consist of diffuse or circumscribed varicose dilatations of the hemorrhoidal veins, lying either in the subcutaneous tissue of the anus external to the sphincter (external hemorrhoids) or in the submucous tissue of the lower portion of the rectum (internal hemorrhoids).

The hemorrhoidal veins surround the lower portion of the rectum and *there form the hemorrhoidal plexus.* The majority of these veins enter into the inferior or external inferior hemorrhoidal veins, and from there into the common pudic and iliac veins; others pass into the median inferior hemorrhoidal veins and so into the internal iliac vein and the inferior vena cava. A small number of the veins enter the superior hemorrhoidal veins, thence into the inferior mesenteric veins and the portal system.

In portal obstruction blood from the hemorrhoidal plexus can pass into the vena cava; while back pressure on the vena cava inferior, when of cardiac origin, may affect the hemorrhoidal system. All hemorrhoidal veins *are devoid of valves.*

**Anatomy.**—External hemorrhoids are visible to the naked eye, lie below the sphincter ani, and are often arranged in groups around the anal orifice. They appear as bluish-red, tortuous vessels encircling the opening; or there may be isolated varicose protrusions, from the size of a pea to a walnut. They may be round, flat, or irregular in shape, and their size sometimes changes in the same patient, often being smaller after defecation.

Internal hemorrhoids often can only be discovered by digital examination or by use of the proctoscope, as they lie above the sphincter. In aggravated cases the patient by bearing down may cause them to protrude. They usually appear as soft nodules of bluish hue, with thin walls.

The diffuse or circumscribed nodular forms may be present, and the last constitute a true varix.

External and internal hemorrhoids may occur together. Piles may occur singly or in pairs, or be multiple and form a ring about the anal opening, both external and internal to the sphincter, and even a third higher ring has been described. In exceptional cases dilated veins are found high up the rectum, even into the sigmoid flexure.

Hemorrhoids are generally believed to be simple venous ectasias and are considered genuine varices, though some maintain them to be true angiomata.

Secondary changes probably account for the various conditions found. The dilated blood-vessels, venous varices, near a group of hemorrhoids may become inflamed, adhere and coalesce, and the walls of the vessels atrophy, so that tumors of some size, resembling cavernous multilocular tumors (angiomata), may develop.

The external covering of the varix may become hard, thick, and resistant, through inflammatory processes. In other cases the walls may become thin and eventually rupture, or the blood coagulate within the pile and form a thrombus.

Phleboliths occur in old cases. A blood-cyst is occasionally formed.

Marked connective-tissue increase may take place in some of the external hemorrhoids; they may present the appearance of skin tabs, or skin externally and mucous membrane internally, which may become edematous and inflamed. They may give rise to warty growths.

The mucous membrane of the rectum near the hemorrhoids is hyperemic, and, with internal hemorrhoids, in a condition of catarrhal inflammation.

Fissure or prolapse of the rectum may be associated with piles.

Allingham has subdivided internal hemorrhoids into three varieties:

1. Capillary piles, resembling nevi, consisting of hypertrophic capillary vessels and spongy connective tissue with thin mucous membrane. They easily bleed.

2. Arterial piles, sessile or pedunculated tumors, glistening or villous, slippery, hard, and vascular.

3. Venous piles, in which the veins predominate. Tumors are large and bluish or livid in color.

Internal piles may be pushed down during defecation. A pedicle may be formed to the tumor and the mass may slip out of the rectum. After defecation they may spontaneously return into the bowel, or can be replaced. If large they may become incarcerated and even gangrenous and slough off. Hemorrhage is a common occurrence.

Inflammatory processes may complicate internal piles, such as ulcers, proctitis, and periproctitis. With the latter abscess may result, which may form fistulæ, external, internal, or complete.

**Etiology.**—Hemorrhoids are frequently met with in practice and the condition is much more common than supposed, as many patients so afflicted never consult the physician. It is rare in children.

The statement is often made that piles occur more frequently in men than in women. Undoubtedly more male cases are found *in our records*. Habitual constipation, pelvic congestion, and sedentary life favor markedly the production of hemorrhoids, and these conditions we find most frequent in women. *Males will at once consult the physician* if there is difficulty in the anal region. Women are, as a rule, extremely sensitive, and usually treat their piles by home remedies until conditions are such as to necessitate advice from the physician. It has been my experience that most of my women patients never refer to " hemorrhoids " unless specifically questioned. Actually, I believe women are the more frequently afflicted.

*Age.*—Piles occur most frequently between thirty and fifty years of age. The modern consensus of opinion is that hemorrhoids are a local disease and not due to any diathesis or faulty state of the general circulation. There are certain peculiarities in the rectal plexus of veins favoring the production of piles which are as follows:

The walls of the hemorrhoidal veins are thin and contain few muscular fibers, and hence their contractile force is less than that of the veins in the lower limbs.

There are no valves in the rectal veins, so that blood can be readily forced back into them. Muscular contraction aids the onward propulsion of the blood in other regions.

In the rectum fecal masses compress the blood-vessels, the sphincteric contractions compress the vessels, and contraction of the abdominal muscles during defecation interferes with the return of blood. They are dependent blood-vessels, and with the unfavorable factors mentioned it is not surprising that hemorrhoids are frequent.

Some of the hemorrhoidal veins enter the portal system, in which there is a low degree of pressure and in which the circulation is readily retarded. Stasis of this system may be a contributory factor in the production of hemorrhoids, as in cirrhosis of the liver; as may also diseases of the heart and lungs leading to engorgement of the venous system.

All conditions which tend to produce interference with the return circulation of blood from the hemorrhoidal vessels, or produce or predispose to hyperemia, influence the production of hemorrhoids. Among such are: chronic constipation; sedentary habits, as in clerks, students, shoemakers, seamstresses, sitting on soft cushions, excessive horseback riding, etc.; enlarged uterus, as from disease or pregnancy; prostatic affections, tumors of the bladder, or growths in the pelvis.

Hemorrhoids may develop suddenly in cases of carcinomatous stricture of the rectum and early in the course of the disease.

Habitual constipation favors the production of piles. Hemorrhoids also may be the primary condition, and on account of the pain of defecation the patient avoids having a movement as long as possible. Excessive venery is given as causing congestion in these regions. Abuse of alcohol, spiced food, fatty food, excessive quantities of food and drink are believed by some to favor the plethoric habit and produce fulness of the portal circulation, with resulting piles. Gourmands of this type are often of sedentary habit and are frequently disposed to constipation—a more likely cause. Von

Recklinghausen has shown that piles are by no means common in plethoric individuals, but more in those of lean habit, of poor muscular development, with defective metabolism, who lead a sedentary life, and who are anemic, with poor circulation. Catarrh of the rectum is given as a cause, but it is not always easy to determine whether the catarrh or piles were primary. Drastic purgatives are said to produce hemorrhoids, but they are used for the constipation, which may be the chief factor. Cases do occur in which it seems probable that the excessive use of purges has produced hemorrhoids with prolapse.

Among the Orientals, who lead a sedentary life, hemorrhoids seem quite common. Hereditary anatomic peculiarities have been suggested as a cause, since piles are often found in several successive generations.

**Symptoms.**—*External Hemorrhoids.*—These are chiefly of local character. The patient may have a sensation of fulness, clogging, or pulsation in the lower rectum. At times there may be a feeling of obstruction at the time of bowel movement. Constipation usually precedes the attack.

There is itching of the anus and perineum. The anus may be tender and swollen, and if the buttocks are drawn apart external piles, single or in clumps, round and bluish in color, distended with blood, are present. Stains of blood are often found on the toilet paper. The exacerbation may quiet down under rational methods. They may become swollen and edematous, painful, and even ulcerate and suppurate, with the production of a fistula.

Tenesmus may be present, and the external pile may be drawn up into the sphincteric circumference and become pinched and strangulated. Under such conditions there are severe pain, throbbing, a desire for defecation with straining, and the patient cannot sit or walk about. Fever, anorexia, and severe constipation accompany the local manifestations.

Hemorrhage is not as marked with external piles.

*Internal Hemorrhoids.*—With internal hemorrhoids hemorrhage is often a prominent symptom. Frequently internal piles can only be detected by digital examination or by inspection through a speculum. They may prolapse and even become strangulated. In such event the pile becomes swollen, turns deep blue, there are agonizing pain, marked tenesmus, occasionally vomiting, constipation, meteorism, fainting, prostration, and fever.

Gangrene and sepsis may occur if the strangulation is not relieved.

With mild types of internal hemorrhoids the only symptom may be an occasional hemorrhage. One must remember that hemorrhoidal veins may be very high up and only be visible with a proctoscope.

Usually, subjective symptoms are present, such as itching, burning, pressure and weight in the anus, and the sensation of a foreign body, with tenesmus. There may be feelings of pressure and even pain in the sacral and lumbar regions. Occasionally soreness over the lower part of the abdomen, pressure in the rectum, bladder, uterus, and vagina; pains radiating to the thighs may be present. Sometimes these symptoms precede a hemorrhage, after which temporary relief occurs.

As a rule, the bowels are irregular and constipated.

Hemorrhages may be frequent, so as to cause marked secondary anemia. Bleeding may be severe and bright red blood gush from the rectum nearly pure, or it may be dark brown mixed with fecal matter. In such an event it lies on the surface of the feces and is not intimately mixed with it. Small quantities of blood are frequently passed with hard stools.

Hemorrhages sometimes occur on no special provocation, or after a horseback or bicycle ride, etc.

Gummy acrid mucus may be discharged from the hemorrhoids due to catarrh of the rectum, and may cause excoriations.

In some the hemorrhage may last for several days and temporary relief may occur. It has been stated that bleeding from hemorrhoids may take the place of menstruation (vicarious).

Dyspnea, palpitation, angina, hiccup, giddiness, despondency, and hypochondria, often attributed to piles, I believe, with Riegel, are merely associated with the constipation present. Proctitis, fissure, and occasionally abscesses or fistulæ may develop.

Disturbances of adjacent organs, such as strangury, hemorrhage from the bladder or vagina, or catarrh of the latter may occasionally occur.

**Diagnosis** is made by inspection and digital examination; occasionally the proctoscope is necessary. The appearance of hemorrhoids has been described.

*Condylomata* encircle the anus and are often present on the scrotum. There are a history of syphilis and manifestations of lues.[1]

Skin tags are whitish looking, do not change their size, and do not bleed when punctured. Piles differ in the last regard.

Internal hemorrhoids are reddish blue and bleed when punctured; polypi do not do so. The latter occur most frequently in children.

Carcinoma presents a hard consistency, and produces stenotic symptoms and cachexia. A small section placed under the microscope will settle the diagnosis.

Piles may occur suddenly in association with cancer.

**Prognosis.**—Piles are frequently a chronic affection and may exist throughout life. In cases occurring during pregnancy, restitution to normal is possible. This is true of the milder types if the cause is corrected. They may markedly diminish in size and then an exacerbation take place. They rarely endanger life unless strangulation with gangrene or a large abscess or dangerous hemorrhage occur.

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[1] The Wassermann or Noguchi reaction should be tested.

**Treatment.**—Excesses of all kinds in food, drink, and venery should be avoided. The patient should lead an out-of-door life, take proper exercise, and have a daily soft evacuation of the bowels.

All conditions causing venous engorgement of the rectum should be avoided, such as constant standing or sitting, horseback and bicycle riding. The patient should not sit on warm soft cushions, but on cane-seats or those covered with leather or horse-hair.

*Diet.*—The diet should be mixed in character and in part consist of a considerable variety of green vegetables and raw, ripe and cooked fruits, to regulate the bowels.

It should suit the individual, and a stout full-blooded person must be somewhat limited in his diet. The patient should not take three large meals at long intervals, but preferably smaller and more frequent feedings.

Alcoholic beverages, strong coffee, strong tea, highly seasoned dishes, cheese, coarse brown bread, peas, corn, and baked or lima beans are best avoided.

String beans, spinach, asparagus, and green salads aid bowel action. Potatoes, rice, beets, and cauliflower may be allowed in small quantity. Raw fruits, such as apples, pears, prunes, grapes, oranges, and stewed fruits, such as prunes, baked apples, etc., are of value. Soups of various kinds are allowed. Hot breads, richly spiced foods, and rich desserts should be avoided.

Matzoon, koumiss, bacillac, lactone-milk, and buttermilk often have an excellent effect on the bowels. They can be taken as the extra meals midway between the usual meals. A few crackers or zwieback with plenty of butter can be given. Some recommend water only between meals and interdict much of that. Unquestionably a certain amount of fluid with the food is an aid to digestion and bowel action. I have seen severe cases of constipation result from the elimination of all fluids at meals.

At least 8 ounces (250 c.c.) of fluid in the form of broth, soup, cocoa, or water should be taken at each meal. On rising, the administration of a glass of water often aids bowel action, and between meals an equal amount should be given, or matzoon or koumiss substituted.

Outdoor exercise, such as walking, is of service. It should not be carried to the point of fatigue, and is contra-indicated during acute inflammation of the piles or if hemorrhage or unpleasant symptoms result. Massage, gymnastics, and Swedish movements are of value.

*Bowels.*—Constipation should receive appropriate treatment. *Powerful cathartics should be avoided.* The dietary methods mentioned should be carried out. The patient should go to stool daily at a definite hour. I have found the administration of olive oil, 1 to 2 tablespoonfuls (15.0–30.0) t. i. d. before meals, an aid to bowel action. The injection of 1 to 2 ounces (30.0–60.0) of olive oil into the rectum with a small soft-rubber ear syringe just before having or attempting to have a movement is a valuable procedure. It also renders the passage easier and less painful.

Aloes should be avoided in hemorrhoid cases.

Among our simple remedies are compound liquorice powder, 1 dram (4.0); fluidextract of cascara or aromatic fluidextract, 1 dram (4.0); extract of cascara, 1 to 5 grains (0.06–0.3); purgen (phenolphthalein), 1½ to 5 grains (0.1–0.3); tincture of rhubarb, 1 dram (4.0), or extract of rhei, 4 grains (0.25), and regulin. Other remedies are described in the chapter on Constipation.

Carlsbad, Kissengen, and Saratoga waters are of service, especially at the springs.

A good prescription is the following:

℞. Ext. cascara.......................... gr. xv (1.0);
Ext. belladonnæ }
Ext. nucis vomicæ } ..................āā gr. iij (0.2).—M.
Make 12 pills.
Sig.—One to two at bedtime.

*Hygiene of the Anus.*—After defecation, cleansing with a moist sponge or wet cotton is less irritating than paper.

External piles should be protected with a small piece of cotton moistened with sweet oil or covered on the anal side with vaselin, boric acid, or zinc ointment. Cold sitz-baths and cold ablutions to the rectum are valuable. An external douche with cold water is excellent, thus: A fountain syringe is filled with cold water at about 60° F. and several ounces of witch-hazel added, or a hot douche at 110° to 115° F. can be substituted. The rectal tip is placed close to the anus, which is then thoroughly douched. This is especially applicable for external piles, and lessens congestion even when they are internal.

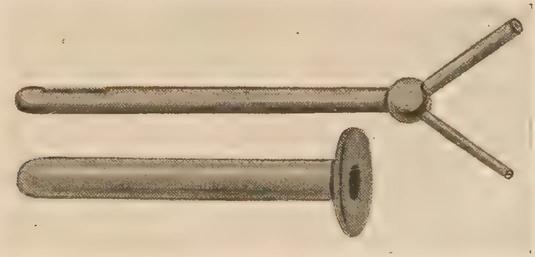

Fig. 267.—Kemp's tube (cooler) for hemorrhoids or prostate.
Fig. 268.—Kemp's ice tube for hemorrhoids.

For external inflamed piles stronger medicated local applications can be employed than with internal piles, where greater absorption occurs.

*External Hemorrhoids.*—With inflamed piles absolute rest in bed or on a couch should be enjoined. A small ice-bag or gauze wrung out in cold solution of witch-hazel and water, equal parts, or of lead-and-opium wash, are excellent applications. Some prefer the application of warmth; cold is usually more efficacious.

For the application of cold to relieve congestion, both to external and internal hemorrhoids, the simple instruments in Figs. 267 and 268 are of value.

The ice tubes for hemorrhoids are made in a nest containing several sizes, in appearance like a very small test-tube with a broad flange, to prevent slipping into the bowel. The tube is filled with powdered ice, corked, well lubricated, and inserted into the anus. They are made of glass or metal, the latter the safer.

The prostatic cooler is of small caliber—a simple closed tube with a large entering and small return attachment. The fountain syringe is attached to the large branch and a soft outflow tube to the smaller. Very cold (preferably) or hot water can be employed. The soft-rubber rectal bag (Fig. 269) is made on similar principles, but is more bulky. A small rubber condom, with two catheters tied in, one for the entering and the other for the outflow current, can be substituted for this bag. They were reported some years ago.[1]

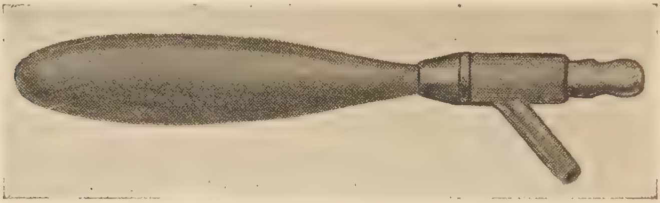

Fig. 269.—Kemp's soft-rubber rectal bag, used as a cooler for hemorrhoids.

In addition, one can attach a small catheter to the tip of the fountain syringe and, inserting the soft tube about 1½ inches, thus douche the internal hemorrhoids. The current should be slow, the patient evacuating the fluid around the catheter every minute or so. Hot or cold normal saline solution with witch-hazel, 1 ounce (30.0) to the pint (500 c.c.), can be employed. The old Bodenhamer recurrent tip or the flexible or hard-rubber recurrent tubes (Kemp) can be used, introducing them only about 2 inches; but with inflamed piles they are apt to cause more pain than the small soft catheter.

I have found the following prescriptions, recommended by Samuel Gant, of value for the relief of pain and inflammation in external hemorrhoids:

| | | | | |
|---|---|---|---|---|
| ℞. | Liq. plumbi subacet.................... | ʒiv | 15 | |
| | Tinct. opii.............................. | ʒiiss | 10 | |
| | Aq. destil.........................q. s. | ℥iv | 125 | .—M. |

Sig.—Apply cold, on gauze.

---

[1] Manual on Enteroclysis, Hypodermoclysis, and Infusion, 1900.

| | | | | | |
|---|---|---|---|---|---|
| ℞. | Ung. stramonii | ʒiss | 6 | | |
| | Ung. belladonnæ | ʒiiss | 10 | | |
| | Ung. acidi tannici | ℥ss | 15 | | .—M. |

or

| | | | | | |
|---|---|---|---|---|---|
| ℞. | Morph. sulph. | gr. iij | | 195 | |
| | Cocainæ hydrochlor. | gr. xij | | 78 | |
| | Vaselin | ℥ij | 60 | | .—M. |

or

| | | | | | |
|---|---|---|---|---|---|
| ℞. | Cocainæ hydrochlor. | gr. v | | 325 | |
| | Ext. bellad.<br>Ext. opii<br>Ext. aconite<br>Ext. stramonii | āā ʒij | 8 | | |
| | Glycerin | ʒss | 2 | | .—M. |

Sig.—Apply on cotton, externally.

This last prescription I prefer to use first at about one-third or one-half strength. It should be employed for only the temporary relief of severe pain and should be left on only for a short period, about fifteen minutes.

Cold or, at times, hot injections, and opium and belladonna suppositories are excellent for tenesmus.

For internal hemorrhoids, when prolapsed, inflamed, and difficult of reduction, it is advisable to apply cold, such as ice-water to which witch-hazel has been added, ice, or lead-and-opium lotion (cold), for a brief period, to lessen edema and inflammation.

Local *application of adrenalin* (1:1000) *to an irreducible pile* lessens congestion and aids reduction.

One of the following ointments is excellent as a sedative:

| | | | | | |
|---|---|---|---|---|---|
| ℞. | Morph. sulph. | gr. viij | | 52 | |
| | Hydrarg. chlor. mit. | gr. xij | | 78 | |
| | Vaselin | ℥j | 30 | | .—M. |

(Gant.)

| | | | | | |
|---|---|---|---|---|---|
| ℞. | Ext. opii | ʒss | 2 | | |
| | Cocainæ hydrochlor. | gr. x | | 65 | |
| | Mentholi | gr. xx | 1 | 30 | |
| | Ung. zinci oxidi | ℥j | 30 | | .—M. |

The prolapsed hemorrhoid should be well lubricated before the physician endeavors to push it up with the finger. Occasionally an anesthetic may have to be administered. There are the dangers of strangulation with gangrene and sepsis from an unreduced hemorrhoid.

For ulcerated hemorrhoids the following are useful:

| | | | | | |
|---|---|---|---|---|---|
| ℞. | Bismuth subnit. | ʒij | 8 | | |
| | Hydrarg. chlor. mit. | ℈ij | 2 | 6 | |
| | Morph. sulph. | gr. iij | | 195 | |
| | Glycerin | ʒij | 8 | | |
| | Vaselin | ℥j | 30 | | .—M. |

Sig.—Apply with a pile syringe (Allingham).

| | | | | | |
|---|---|---|---|---|---|
| ℞. | Cocainæ mur. | gr. xij | | 78 | |
| | Iodoformi | ʒj | 4 | | |
| | Ext. opii | ʒss | 2 | | |
| | Vaselin | ℥j | 30 | | .—M. |

Sig.—Apply with a pile syringe (Mathews).

For hemorrhage the following are of service: Rectal injections of ice-water, 8 ounces (250 c.c.), alone or with tannic acid, 5 grains (0.3), dissolved therein, or alum, 1 dram (4.0), or witch-hazel, equal parts; local application of ice-bags or the ice tube.

Several ounces of Trémolière's solution—gelatin (5 per cent.) with chlorid of calcium (2 per cent.)—can be injected into the rectum. It was originally used as a local styptic.

If the bleeding is from external piles, styptics can be applied in ointment form; if from internal hemorrhoids, the ointment can be inserted with the finger or applicator, or as a suppository.

Among such useful remedies are unguentum acidi tannici and unguentum gallæ:

| | | | | |
|---|---|---|---|---|
| ℞. | Unguentum acidi tannici | ʒiv | 16 | |
| | Unguentum stramon.<br>Unguentum belladonnæ | āā ℥j | 30 | .—M. |

Ft. ung.

| | | | | |
|---|---|---|---|---|
| ℞. | Suprarenal ext. | gr. v | | 3 |
| | Ol. theob. | gr. xxx | 2 | .—M. |

Ft. suppos. No. 1.

| | | |
|---|---|---|
| ℞. | Adrenalin chlorid (1: 1000) | ♏x (0.592) |
| | Ol. theob. | q. s.—M. |

One suppository.

| | | | | |
|---|---|---|---|---|
| ℞. | Ichthyol<br>Acidi tannici | āā gr. v | | 3 |
| | Ext. belladonnæ<br>Ext. stramon. | āā gr. $\frac{1}{3}$ | | 022 |
| | Ext. hamamelis | gr. x | | 6 |
| | Ol. theob. | q. s. | | .—M. |

Ft. one suppository. (J. P. Tuttle.)

An ointment or suppository containing chrysarobin has been advised for the treatment of hemorrhage from piles:

| | | | | |
|---|---|---|---|---|
| ℞. | Chrysarobin | gr. xv | 1 | |
| | Ext. belladonnæ | gr. v | | 3 |
| | Iodoform | gr. x | | 6 |
| | Petrolati | ʒss | 15 | .—M. |

Ft. unguentum.

| | | | | |
|---|---|---|---|---|
| ℞. | Chrysarobin | gr. ij | | 13 |
| | Acidi tannici | gr. iij | | 194 |
| | Iodoformi | gr. ij | | 13 |
| | Ol. theob. | gr. xxx | 2 | .—M. |

Ft. one suppository.

In many cases of hemorrhage the simpler remedies are sufficient. Rarely it may be necessary to tampon the rectum. This can be performed by slipping in a piece of cheese-cloth like the finger of a glove, in effect a bag, with the outside well lubricated. This is packed with cotton tampons or strips of gauze, and the distended bag drawn down against the sphincter. It can be done in emergency without an anesthetic. If these measures fail, it may be necessary to ligate the bleeding artery or, if this be impossible, then the entire hemorrhoid.

**Radical Treatment.**—*Dilatation of the Sphincters.*—By means of this the spasm of the sphincter is stopped, the pressure on the blood-vessels is relieved, and the bowels act more easily. It will frequently be of benefit in the early stages of hemorrhoids, and I have seen it relieve severe symptoms in advanced cases, especially if fissure is associated. The author has noted cases in which several years after dilatation the patient has claimed to be in comfort, with no recurrence. As a palliative the procedure is justifiable, especially if fissure is present.

Gradual dilatation by the use of specula of increasing size or of dilators can be carried out. The procedure takes several weeks and is necessarily very painful. If this method is carried out, air dilatation with Robert's dilator (Fig. 270), made on the principle of Barnes' cervical dilator bag, is the most satisfactory. The hard-rubber dilators cause more pain.

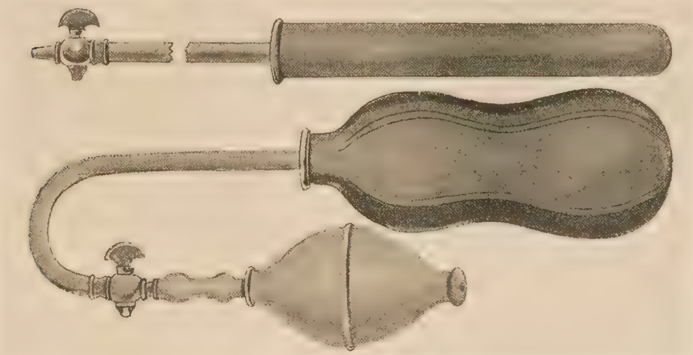

Fig. 270.—Robert's dilator.

Rapid and complete dilatation under anesthesia is the best method when safe for the patient. With the aid of a skilled anesthetist it can be satisfactorily performed under nitrous oxid. Other anesthetics can be employed. The thumbs must be inserted into the rectum and the sphincter *dilated gradually*, both laterally and anteroposteriorly. All spasm must be overcome and the sphincter thoroughly relaxed. Violence must not be employed. An opium suppository, 1 grain (0.065), with belladonna, $\frac{1}{3}$ grain (0.02), should be inserted and the bowels kept closed for about forty-eight hours.

*Carbolic Acid Injection.*—This method has been recommended in order to produce shrinking of the piles and so avoid radical operation. It should not be performed if the hemorrhoids are inflamed.

The following solutions for injection have been recommended:

| | | | |
|---|---|---|---|
| ℞. | Acid. carbolic. (Calvert's) | ʒij | 8 |
| | Acid. salicylic. | ʒss | 2 |
| | Sod. bibor. | ʒj | 4 |
| | Glycerin (sterile) q. s. | ℥j | 30.—M. |

(J. P. Tuttle.)

The solution should be syrupy and clear; if white, it is imperfect.

The average injection into a hemorrhoid is 5 minims (0.296). Not more than 10 minims (0.592) should be injected into two to three hemorrhoids. After the injection a suppository of—

| | | | |
|---|---|---|---|
| ℞. | Opii (pulv.) | gr. j | 065 |
| | Ext. belladonnæ | gr. $\frac{1}{3}$ | 022 |
| | Iodoformi | gr. ij | 13 |
| | Ol. theob. | q. s. | .—M. |

Carbolic acid and glycerin, equal parts; or carbolic, 1; glycerin, 3; water (distilled), 3, have been employed.

Inject 3 to 5 drops with a hypodermic syringe into the center of the pile. Care must be taken that none of the solution drops from the needle and cauterizes the mucous membrane. An enema is first given, the piles cleansed and dried, and then anointed with iodoform ointment after injection with carbolic.

A few drops of a 1 per cent. cocain solution may be first used subcutaneously to deaden the pain.

It is preferable to inject one hemorrhoid at a time. Considerable pain at times follows. The possible dangers are ulcer, abscess, and fistulæ. There is some danger of sepsis and cases have been reported.

*Cauterization with Fuming Nitric Acid.*—The surrounding parts are covered with a thick layer of vaselin, except the pile that is to be treated. This is painted with the nitric acid by means of a small stick or glass rod. Care must be taken that the acid touches no other spot. The pile turns a grayish-green color as a result. Iodoform ointment or vaselin is applied and the hemorrhoid pushed back into the rectum. Allingham advises carbolic acid as a substitute for the nitric acid. There is an element of danger from sepsis in this method.

*Ligature* of hemorrhoids under anesthesia without removal and then allowing them to *slough off* has been employed. The method is not to be recommended for obvious reasons.

*Crushing.*—The hemorrhoid is crushed longitudinally by means of a special instrument, the projecting portion *cut off*, and pressure kept up for about a minute. This technic is not advised.

*Clamp and Cautery.*—Each pile is seized with a volsellum forceps and drawn well down. The clamp is applied to embrace its base. The portion above the clamp is cut off with scissors, and the cautery iron, heated to a dull red, is applied to the stump until the vessels are well seared. Either the thermocautery or galvanocautery can be employed. Excellent results have been secured by this operation.

*Ligature and Extirpation* (*Allingham's Operation*).—Incision is performed at the base of each pile through the mucosa, and the pedicle dissected out. This is ligatured and the pile cut off with scissors. Some operators remove every alternate hemorrhoid, the rest usually shrinking as a result of operation. Others incise the mucous membrane above the base of the pedicle and force it back like a cuff. After ligature and removal of the pile, the cuff of mucosa is drawn down and a single stitch taken. This last method is to be recommended. There is less danger of postoperative hemorrhage, and healing is more rapid.

*Extirpation and Suture* (*Whitehead's Operation*).—The mucous membrane containing the hemorrhoids is dissected out, the incision frequently encircling the anus. The piles are removed, and the mucosa, being loosened up, is drawn down and attached by sutures to the skin at the anal margin. *Stricture* or incontinence of feces are sequels which may follow this operation. Subsequent treatment by bougies is necessary if stricturing be feared or if it occur.

In these operations anesthesia is necessary, and dilatation of the sphincter previous to removal of the hemorrhoids.

It is usually customary to close the bowels for twenty-four to forty-eight hours after operation by means of opium suppositories, though Graser opens the bowels at once.

*Gant's Water Infiltration Method*.—This method, devised by Samuel Gant, is of special value in cases where an anesthetic is contraindicated or when the patient objects to its employment. With a large hypodermic, sterile water is injected about the base of each hemorrhoid in the submucosa, producing thorough infiltration. An incision is then made around the base of the pile, the pedicle dissected out, the latter ligated, and the pile cut off with scissors.

Careful preliminary dilatation of the sphincter is carried out.

Having seen this operation performed by its originator and also the ultimate results, I can recommend it as excellent.

**Complications of Hemorrhoids.**—*Prolapse of the Rectum.*—*Etiology.*—Rectal prolapse is a fairly frequent complication of hemorrhoids, though it may occur alone. It may involve the mucous membrane alone or all the coats of the rectum (procidentia recti). Among other causes of prolapse are polypi, vegetations, tumors; conditions tending to weaken the sphincters, such as ulceration or operative incisions, spinal paralysis, or traumatism; conditions producing muscular spasm, such as worms, dysentery, phimosis, cystitis, calculus, enlarged prostate, or stricture of the urethra.

In some cases with hemorrhoids, the prolapse may be only partial as regards the circumference of the rectum, while in others it involves the entire circumference and there is a scarlet or livid mass projecting from the anus. An internal prolapse of the rectum may occur, in which the upper part descends through the lower part, but does not appear outside the anus. It is probably due to relaxation of the ligaments of the rectum. It corresponds rather to an intussusception.

Weakness and paralysis of the sphincters are predisposing factors.

Prolapse of the rectum is frequent in debilitated children, especially if there is intestinal catarrh with a tendency to frequent stools accompanied by straining. Rectal prolapse occurs in elderly people. also in cases of severe constipation, and as a result of frequent pregnancies.

*Symptoms.*—With moderate prolapse, during the act of defecation, a protrusion of the rectum from 1 to 2 inches long appears outside the anus. It is red or bluish red in color and puckered in appearance, covered with some mucus. The swelling is continuous, with the skin on one side and with the mucous membrane on the other, and is arranged in folds which radiate from the central aperture toward the circumference.

In advanced cases the mass resembles a tumor with a star-like opening at its center, and the color is a paler red or bluish. With children the mass generally protrudes only when at stool; while with adults it comes down more readily or often remains constantly down. At first it is spontaneously reducible, later, easily replaced by slight pressure. and finally it may become very difficult or nearly impossible of reduction.

It often becomes inflamed or ulcerated, and in old cases incontinence of feces may occur.

At first there is no or little pain, but after a time it may become quite severe.

There is a tendency for the prolapse to increase in size. If inflammation occur, there will be fever and constitutional disturbances.

As a rule, there is no marked bleeding from the prolapse itself, but more of an oozing. Hemorrhage from associated hemorrhoids may occur.

*Diagnosis.*—This is made from the appearances described. If operation is contemplated, it is important to differentiate between prolapse of the mucosa alone or of the rectum. In the latter event the peritoneum may be involved and the intestines be found in the prolapsed portion.

Prolapse of the mucosa is not as firm and thick to the feel, the folds of the mucous membrane radiate from the orifice to the circumference, and the opening is circular and patulous.

With prolapse of the entire wall the tumor is more conic, the walls are thick and firm, the orifice is slit-like. When the mass is pressed between the fingers, gurgling of gas in a contained loop of gut may sometimes be heard and resonance obtained on percussion.

In some cases where there is much inflammatory thickening it is difficult to differentiate. In following out treatment this possible danger should be recognized and due precaution be exercised.

Internal prolapse is more difficult to recognize. Digital examination of the rectum is necessary. First keep the finger close to the anterior wall until it passes into a cul-de-sac. Withdraw it slightly and examine the center of the mass until an orifice is found into which the finger or a bougie can be passed for some inches into the rectum.

It may be necessary for the patient to bear down during examination.

*Treatment.*—Predisposing factors should be eliminated, hemorrhoids, polypi, etc., removed. Cleanliness after defecation should be observed. The prolapse should be replaced with the patient preferably in the knee-chest position. The mass should be thoroughly lubricated with vaselin and gently pressed into the rectum. If a considerable portion is down, a large flexible bougie can be cautiously passed into the bowel, pushing before it the upper part of the descended gut, and pressure (taxis) should be employed evenly on the other surrounding portions with the fingers.

When the prolapse occurs frequently, a rectal supporter should be worn, such as a soft-rubber ball attached to the anus by means of a belt and T-bandage. A supporter can be improvised by employing a small roll of gauze covered with oiled silk and attaching it to a home-made T-bandage.

With children palliative treatment is more successful. The cause of the difficulty should be investigated and removed if possible, such as a polypus. The general health should be built up, mild laxatives should be administered, and the diet regulated to secure easy bowel action if there is constipation. If diarrhea is a factor, it should receive treatment. The child should be instructed not to strain at stool, and in many cases it is wise to have him defecate in the *knee-chest* posture over a vessel to prevent prolapse as much as possible, or in the Sims posture, even at the expense of soiling the bedding or clothing, which can be easily changed.

If prolapse occurs, it should be washed with cold water or weak boric acid solution or an astringent alum solution, 1 dram (4.0) alum to 8 ounces (250 c.c.) water, smeared with vaselin, and gently returned into the anus. A weak zinc sulphate solution can be substituted. After this the patient should remain in the recumbent position for about half an hour, preferably lying on the abdomen.

Radical measures must frequently be adopted. Cauterization of the prolapsed portion with fuming nitric acid or acid nitrate of mercury, as suggested by Allingham, or (preferably) with the thermocautery under anesthesia, has benefited or even cured cases. After cauterization the prolapsed part should be lubricated and returned into the anus. Strictures are occasionally produced by this procedure.

Other methods have proved successful, such as excision of elliptic portions of the mucous membrane with subsequent suture; extirpation of the prolapsed portion; revision or narrowing of the caliber of the rectum; suspension of the prolapsed rectum by attaching its mesentery to the abdominal wall; or suspension of the lower end of the sigmoid flexure.

In some cases Gant infiltrates the mucosa with water or a 1 per cent. eucain solution at a number of points until glassy white welts are formed. These are in turn seized, pulled down, ligated, and left to slough off. The rectum is shortened and narrowed as healing of the ulcerated areas takes place.

*Fissure of the Anus.*—This condition quite frequently occurs with hemorrhoids. The fissure consists of an oblong tear of the mucous membrane of the anus, which gives rise to pain and spasmodic contraction of the sphincters. Some fissures are slight abrasions or they may be fairly large and deep. The edges may appear healthy or be inflamed or indurated. Those of longer duration present the appearance of an ulcer. In fact, they are classified as irritable ulcer.

Anal fissure is usually caused by an injury or tearing of the mucosa at the edge of the anus. Excessive straining or the passage of dry hard scybalæ are factors. It is more common among women. The fissure may occur in any location, though most frequently at the posterior portion of the anus. It generally lies parallel and close to the external sphincter, though it may be higher up near the internal sphincter or above it.

*Symptoms.*—Severe pain in the rectum during defecation and at times tenesmus, persisting for a time thereafter, are present. The pain may be very severe in character, so that the patient dreads to have a stool, and increased constipation results.

A small fissure over the external sphincter usually causes greater disturbance than a larger one higher up. There may be reflex irritation of the bladder and a discharge of pus and blood from the rectum. From the pain and suffering the nervous system may become affected.

*Diagnosis.*—This is made from the symptoms and by local examination. The patient should lie on the left side and be told to bear down. On opening the anus with the finger and thumb, a fissure (crack) or small club-shaped ulcer can frequently be seen. It may be red and inflamed or, if more chronic, of a gray color with indurated edges. The pain may be so severe that before local examination it may be necessary to introduce a suppository of cocain, $\frac{1}{4}$ grain (0.016). Rarely an anesthetic may be required.

For a fissure higher up, examination with the speculum may be necessary.

*Treatment.*—Hemorrhoids or polypi, if present, should be removed. The dilatation of the sphincter performed during this operation and removal of the cause of the fissure usually will cure the case. An incision through the base of the ulcer can be made at the same operation as a precaution.

Recent fissures are at times cured without operation. The patient should be in the recumbent position, hot lead-and-opium lotion can be applied externally to the anus if there is much pain, as heat relieves pain and spasm.

The following ointment and suppository are of value for the same purpose:

| | | | | |
|---|---|---|---|---|
| ℞. | Ext. conii | ʒij | 8 | |
| | Olei ricini | ʒiij | 12 | |
| | Ung. lanol. q. s. | ℥ij | 60 | .—M. |

Ft. unguentum.

| | | | | |
|---|---|---|---|---|
| ℞. | Ext. belladonnæ | gr. ⅓ | 02 | |
| | Ext. opii | gr. ½ | 03 | |
| | Ol. theob. | gr. xv | 1 | .—M. |

One suppository.

Locally, the fissure can be touched with silver nitrate solution (I have used the pure silver nitrate stick twice a week), varying in strength from 10 to 30 grains (0.6–2.0) to the ounce (30.0) of water every two to three days; on the alternating days a 5 per cent. cocain solution can be carefully applied, only a few drops; or the following ointments can be alternated with the silver application, or substituted for a time:

| | | | | |
|---|---|---|---|---|
| ℞. | Ichthyol | gr. xxx | 2 | |
| | Lanolin | ʒij | 8 | |
| | Petrolati q. s. | ℥j | 30 | .—M. |

Ft. unguentum.

or

| | | | | |
|---|---|---|---|---|
| ℞. | Hydrarg. chlor. mit | gr. xv | 1 | |
| | Pulv. opii<br>Ext. belladonnæ āā | gr. v | 3 | |
| | Petrolati | ℥ss | 15 | .—M. |

Ft. unguentum.

Gradual dilatation of the sphincter without anesthesia, as described under Hemorrhoids, especially with Robert's dilator, is sometimes employed, but this procedure is painful.

Dilatation of the sphincter under anesthesia is *often found curative of obstinate cases.* Nitrous oxid is a valuable anesthetic for this purpose.

Some recommend infiltration of the fissure with a hypodermic of 1 per cent. cocain, and a free incision through its base to the sphincters. Gant anesthetizes the tissues posterior to the rent by injecting them with sterile water or a 0.125 per cent. eucain solution until they become white. He then severs the sphincter with scissors or a bistoury.

An incision of the fissure under anesthesia, combined with sphincter dilatation, may be required.

*Proctitis.*—If proctitis is present with hemorrhoids, it must receive appropriate treatment, as described under the section on that subject.

## CHAPTER XXX

# APPENDICITIS

(*Synonyms.*—Inflammation of the Vermiform Appendix; Perityphlitis; Appendicular Inflammation; Scolecoiditis.)

Inflammatory conditions involving the right iliac region have been called by a variety of names: Iliac phlegmon; typhlitis (inflammation of the cecum); perityphlitis (inflammation of the covering of the cecum); and paratyphlitis (inflammation of the retroperitoneal tissue behind the cecum). Though typhlitis was considered to be the cause of most of the inflammations in the right iliac fossa, modern investigation has demonstrated that appendicitis is the chief factor.

Some go so far as to state that even stercoral typhlitis (an inflammation due to fecal accumulation) *does not exist.* This is an error, as C. A. McWilliams,[1] though admitting it to be rare, shows that an acute or chronic primary typhlitis may occur independently of appendicitis, dysentery, tuberculosis, actinomycosis, or cancer; and that it may be either idiopathic in origin or due to coprostasis.

Autopsy reports and findings at operations have confirmed this, "a typhlitis" with the formation of perityphlitic abscess or general peritonitis, while the *appendix remains normal.* Howard Kelly has reported 14 cases presenting primary lesions in the cecum, the *appendix being normal.*

Over 90 per cent. of inflammations are due to the appendix; the rest to the cecum.

The symptoms of typhlitis are usually indistinguishable from those of appendicitis and the indications for operation are the same. The literature on appendicitis is enormous, and I shall only mention the names of a few of those specially identified with the operative technic and investigation of appendicitis, namely: Reginald Fitz, Richardson, Sands, McBurney, Bryant, Bull, Weir, Fowler, Hartley, Mayo, Dawbarn, R. T. Morris, Wyeth, Blake, Brewer, Hotchkiss, Deaver, and Howard Kelly.

**Position of the Appendix.**—It takes its origin generally from the posterior and median surface of the cecum, corresponding to McBurney's point (1½ inches from the anterosuperior spine of the ileum), on a line drawn from the spine to the umbilicus. The average length is from 3 to 5 inches, but it may vary markedly. The statistics as to its direction are quite variable. It may point downward and inward, downward behind the cecum, or upward, upward and inward, transversely inward, or outward. In many cases the appendix is quite long, and the position and length thus explain the variable locations of adhesions or abscess. It may, therefore, come in contact with the male bladder or rectum, with the uterus or right tube and ovary, or even the left tube, or with the small intestine, or it may pass up as high as the liver or right kidney, even to the left rectus, and close to the spleen. It has been found behind the peritoneum with no peritoneal covering. Bryant has reported it outside the peritoneal cavity in 3 cases. It has been found quite frequently in hernial sacs, and on several occasions in the scrotum. It often hangs free in the abdominal cavity. In a few instances intussusception of the appendix into the cecum has been reported, the appendix being apparently absent, yet the patient suffering from symptoms. These are described under Intussusception as Intussusception of the Appendix. Moreover, the appendix may be beneath the serous covering of the cecum, so that it cannot be determined by palpation. An incision through the serosa will reveal it. This condition undoubtedly accounts for the so-called congenital absence of the appendix.

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[1] Ann. of Surg., June, 1907.

**Peculiarities of the Appendix.**—The lumen of the canal is extremely narrow; the organ is bottle shaped, the narrowest part being near the entrance into the cecum. Gerlach's valve, a reduplication of the mucous membrane of the appendix near its origin from the cecum, makes the entrance of material more difficult, and also its exit. The circular muscular fibers are somewhat scanty. These factors tend to produce stagnation and predispose to infection.

There are many lymph-follicles (adenoid tissue) in the appendix, which usually persist up to the age of about thirty and then begin to retrograde, so that infection is easy, as in the tonsils. In many cases the appendix is completely surrounded with peritoneum, in some it is only partially covered, and the uncovered portion is in direct contact with the retroperitoneal connective tissue, which readily accounts for the so-called perityphlitic abscess. The appendix has a mesentery of its own, as a rule, the meso-appendix, but it is not constantly present. It rarely reaches up to the tip, but usually only one-half to two-thirds of the distance. It is believed to have some influence on the shape of the appendix, as when it is relatively short the latter may be bent. Crile,[1] in an analysis of 1000 cases of appendicitis, found that in a large percentage there was a short meso-appendix, causing fixation of the proximal part and leaving the distal end free, which tended to cause the appendix to fold on itself and interfered with the circulation (an anatomic angulation).

The blood-supply of the appendix is quite scanty. The vessels (the appendicular and a few cecal branches) Fowler believes to be functionally nearly end arteries, the most abundant being from the vessels (the appendicular) in the meso-appendix, so that the vascular supply of the tip of the organ is poor.

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[1] Ohio State Med. Jour., June, 1907.

In females, Clado[1] holds that there is a third source of blood-supply from a vessel passing through the appendiculo-ovarian ligament (a fold of peritoneum passing from the meso-appendix to the broad ligament), and this possibly explains why appendicitis is less common in women, on account of the superior vascular supply. Fowler holds that the blood-vessels may be primarily affected. Misplacement and malformation of the appendix may also have an influence. The peculiar anatomic conformation of the appendix may predispose to infection.

**Etiology.**—The chief cause of appendicitis is bacterial invasion, the most common of which present is the Bacterium coli commune. As a rule, the infection is a mixed one, streptococci being frequently associated. The dependence on tube cultures alone is somewhat inaccurate, since the Bacillus coli readily overgrows and destroys less vigorous organisms. Smears should be taken in addition. Careful plate cultures should be made at the time of operation and also cultures under anërobic conditions. The swab should always be brushed against the intestinal walls and not simply dipped in the exudate. In Kelly's 400 cases the Bacillus coli was present in 92 per cent. The proteus vulgaris has been present.

The Diplococcus pneumoniæ, staphylococci, the anaërobes, and the influenza bacillus have been found. These are the most frequent varieties, and are found both in the appendix, appendical abscess, and in the general peritoneum (fluid of the) if peritonitis is present. In isolated cases, appendicitis is believed by some to be a local expression of a general infection, as associated with scarlatina, measles, rotheln, small-pox, chicken-pox, parotitis, influenza, and acute articular rheumatism. It has accompanied suppurative tonsillitis.

Typhoid, dysentery, and, more rarely, tuberculosis may be causes. Actinomycosis has also been demonstrated to have produced it.

Traumatism[2] or injury from lifting have been given as causes, but probably, if appendicitis occurs as a sequel, the organ was previously diseased or damaged, or the symptoms might be due to a circumscribed traumatic peritonitis.

*Constipation* as a factor in the production of appendicitis is a question of dispute. Fitz and Fowler believed that in the majority of patients the bowels acted regularly previous to the attack, and Riegel holds that constipation and diarrhea have no bearing on the subject. Though unquestionably many patients suffering from constipation never suffer from appendicitis, there is one type of case in which it may be a factor, namely, patients in whom there is a tendency to fecal accumulation in the caput coli and lower ileum, with resulting appendix symptoms, probably from circulatory interference, pressure, blocking of the appendix opening, and catarrh secondary to a slight catarrh of the cecum. I have seen several such cases in which there were the typic symptoms, which rapidly subsided after thorough bowel irrigation, ice-bag, and later cathartics, and subsequent treatment of the constipation, with no subsequent attacks during eight or ten years' observation. To this same class belong the rare type of typhlitis due to stercoral ulcer to which McWilliams refers.

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[1] Compt. Rend. Soc. Biol., 1897, vol. iv, p. 133.
[2] Deaver, N. Y. Med. Jour., June 15, 1907.

Dietetic indiscretions, in so far as they are productive of intestinal putrefaction or fermentation with increased bacterial activity, might be a factor in producing an acute attack in an appendix already damaged. Intestinal catarrh, involving the caput coli, in my own experience has been the direct cause of the catarrhal type of appendicitis.

Entozoa, such as the Trichocephalus dispar, Oxyuris vermicularis, Ascaris lumbricoides, tapeworm, and bilharzia, have been factors in the production of appendicitis. An interesting case of perforation of the appendix by a round worm,[1] with appendical abscess resulting, has been reported. Metchnikoff, in 1901, first emphasized the importance of intestinal parasites as the exciting cause of appendicitis, and reported 3 cases in which the feces contained ova of the ascaris and trichocephalus. They all recovered without operation after a vermifuge. Cecil and Buckley report 5 cases of appendicitis produced by the Oxyuris vermicularis at a meeting of the New York Academy of Medicine, May 17, 1910. Movable kidney, through pressure on the mesenteric vein, has been given as a frequent cause by Edebohls. This condition is associated with splanchnoptosis, and the circulatory changes from malposition of the viscera are more probably factors. They occur most frequently with women, yet appendicitis is less frequent in the female.

Foreign bodies entering the appendix, such as *grape-seeds*, *cherry-stones*, *pits*, *pins*, *buttons*, *hairs*, *shot*, *gall-stones*, etc., are rare causes of appendicitis.

Fecal concretions are found frequently and have been mistaken for foreign bodies. Probably normally soft fecal matter enters and is expelled from the appendix. The fecal concretions are generally hard in character from absorption of water and are thickened by mucus. C. B. Lockwood has demonstrated that in many cases they consist of an inspissated mass of bacteria. It is easy to understand how such concretions, if of large size, can exert pressure and even be productive of ulceration, especially if there be any abnormal condition in the appendix. They have been found in one-third to nearly one-half of the cases.

Small concretions might remain in an appendix without harm. They generally lie near the end (tip).

[1] Jour. Amer. Med. Assoc., Sept. 25, 1909.

Diverticula of the appendix may be the cause of appendicitis. Some interesting observations have been made by W. C. MacCarty[1] as to the relations of carcinoma to appendicitis.

1. In a series of 2000 specimens, 0.6 per cent., or about 1 in every 175 appendices removed at operation are malignant.
2. In a series of 5000 specimens, 0.44 per cent., or about 1 in every 225 appendices removed at operation are malignant.
3. In a series of 2000 specimens, 2.2 per cent., or about 1 in every 40 partially or completely obliterated appendices are malignant.
4. In a series of 5000 specimens, 1.6 per cent., or about 1 in every 53 partially or completely obliterated appendices are malignant.
5. Carcinoma of the appendix may occur as early as five years of age and as late as eighty years of age.
6. Of the carcinomata of the appendix found in this series, 77 per cent. *were not capable of being diagnosed from the gross external appearance.*

Harte has only collected 5 cases of primary sarcoma of the appendix, and Powers[2] reports 1 case in a girl of seventeen years.

Williams[3] believes intestinal lithiasis depending on the calcium soaps from fats in food has some bearing on the incidence of appendicitis.

Right tubo-ovarian inflammation may be a factor in the production of appendicitis.

*Age.*—Appendicitis seems most frequent between ten and thirty years, Fitz stating that 50 per cent. occur before the twentieth year, and Einhorn 60 per cent. between sixteen and thirty years. It has been reported as early as the seventh week, but rarely before the third year. At the Presbyterian Hospital, New York, 33.9 per cent. occurred before twenty years, and 68.9 per cent. before thirty years.

*Sex.*—Men suffer from appendicitis much more frequently than women (from 2 to 3 or more to 1). Johnson states 4 to 1; Deaver 64.2 per cent. in males. At the Presbyterian Hospital the percentage was nearly equal.[4]

**Varieties.**—I will describe both the pathologic and clinical types of appendicitis.

From a pathologic standpoint, the following seems the best classification:

1. *Acute catarrhal appendicitis*, in which the mucous membrane is involved, being swollen and edematous, the submucosa is injected with excessive secretion of mucus or mucopus.

This type is mild, and the appendix drains into the cecum with perfect recovery (endo-appendicitis, Fowler).

2. *Acute Diffuse Appendicitis.*—There is inflammation of the

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[1] Jour. Amer. Med. Assoc., Aug. 6, 1910.

[2] N. Y. Med. Jour., Jan. 7, 1911.

[3] Brit. Med. Jour., Dec. 31, 1910.

[4] McWilliams, Analysis of 1417 Operations upon the Appendix, N. Y. State Jour. of Med., March, 1910.

mucosa and thickening of the entire organ, which is rigid, tense, and infiltrated. By some it is classified as catarrhal, but this is incorrect.

The peritoneal surface is hyperemic. There may be erosions or small ulcers and a fecal concretion. There may be mucus or mucopurulent material in the lumen, or in some cases it may be narrowed or obliterated.

This type sometimes hangs free in the abdomen, but more generally is adherent to the adjacent peritoneal structures, and is characterized by the surgeons clinically as *acute non-suppurative appendicitis*. It may resolve without operation.

On the other hand, this pathologic type may be productive of abscess or perforation, when it would be placed under a different class by the surgeons.

3. *Purulent or Suppurative Appendicitis*.—This is a more advanced stage than the former. There is a definite pus-sac formed by the appendix. This type may also perforate and cause local abscess or general peritonitis.

4. *Gangrenous Appendicitis*.—This is characterized by necrosis, local or general. The tip is most frequently involved or the entire organ.

5. *Chronic Appendicitis*.—This may follow the acute, or the process may be slow and gradual from its incipiency. In some cases the changes have been found to be very slight, merely the evidence of a chronic catarrh of the mucosa, probably an extension from a chronic catarrh of the cecum. At times stenosis is present.

On the other hand, the organ has been found firm, slightly enlarged and thickened, the mucosa thickened, and the lumen narrowed. In some cases there may be a stenosis, with formation of a cyst. In others there are concretions or erosions, or partial obliteration of the lumen; or the appendix may become converted into a cord-like structure, embedded in a mass of thick peritoneal adhesions.

6. *Obliterative Appendicitis*.—A gradual involution process occurs in many individuals. The tube is thickened, the peritoneal surface smooth; the distal portion of the lumen may be entirely obliterated, and the organ becomes sclerotic and shrunken. Ribbert found these changes in more than 50 per cent. of subjects over sixty years of age. Normal involution seems to present no symptoms.

On the other hand, W. C. MacCarty,[1] from his observations at St. Mary's Hospital, Rochester, Minn., concludes as follows:

1. Of all appendices removed at operation 23.5 per cent. are partially or completely obliterated.
2. The shortest duration of the process of obliteration when it is continuous is less than ten years.
3. The process may be complete at ten years of age.
4. Obliteration does not occur as a physiologic involutionary process, but is dependent on a definite inflammatory reaction.

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[1] Jour. Amer. Med. Assoc., Aug. 6, 1910.

5. There seemed to be a large percentage of appendices (52 per cent.), with partially or completely obliterated lumina, in association with inflammatory conditions of the gall-bladder (cholecystitis and cholelithiasis). This partial obliteration of the lumen occurred more frequently in females, which may be possible evidence that this inflammatory process in the appendix causes disturbances in the bile passages directly or indirectly, and may have some relation to the fact that cholecystitis is more frequent in females than in males.

MacCarty[1] and McGrath in a subsequent article show that total obliteration of the appendix occurs on the average at 34.6 years, while that of the tip averages at 29.8 years of age. Carcinoma of the appendix occurred 22 times out of 5000 specimens, or in 0.44 per cent. of cases. In 17 cases diagnosis by the surgeon at time of operation was impossible and the carcinomatous condition was only revealed by the microscope. On section there is an orange-colored area in the obliterated portion of the appendix. They hold that all obliterated appendices should be removed and sectioned. Moreover, they found that obliterated appendices may become acutely inflamed and carcinoma may appear at any age in an obliterated appendix. Diverticula of the appendix were found in 17 cases, and of these 56 per cent. occurred in acute appendicitis, and in women. They conclude that all obliterated appendices should be removed.

From a clinical standpoint the physician will find several types of acute and chronic appendicitis, readily deduced from the pathologic classification.

1. *Acute Simple Catarrhal Appendicitis* (*Endo-appendicitis*).—A catarrh of the mucosa of the appendix. This is of mild type and is often secondary to a colitis or to fecal impaction in the cecum. Unless there is occlusion, the inflammatory products usually drain out (non-suppurative type, with complete cure). There may be recurrent attacks.

2. *Acute Non-suppurative Appendicitis* (*Sometimes Incorrectly Characterized as Acute Catarrhal*).—The description is that of the Acute Diffuse Appendicitis, the entire organ being involved.

There is fibrinous exudation agglutinating the appendix to neighboring structures, and the meso-appendix is thickened and inflamed. There may be kinking or torsion of the organ, stricturing, or even obliteration, and a fecal concretion may be left within it.

These cases often escape operation and may not have a recurrence, but there is great liability of the latter. On the other hand, ulceration with perforation may occur.

3. *Chronic Appendicitis*.—This may follow the acute, or the process be slow and gradual from the start. The chronic catarrhal cases may exhibit but little change, or the appendix may be thickened, the mucosa thick and hyperemic, and its lumen narrowed, obliterated, or strictured.

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[1] Surgery, Gynecology, and Obstetrics, March, 1911.

A fecal concretion may remain within it. It may present adhesions. It may follow an acute attack or be chronic from the onset.

These three types are characterized by *absence* of perforation, gangrene, or abscess formation; though type 2 may progress to type 4, with resulting abscess or perforation.

4. *Acute Suppurative Appendicitis* (*Formation of Abscess*).—Of this we have two clinical types, the pathologic *purulent appendicitis* (the appendix distended with pus), on the verge of perforation, or an inflamed appendix enclosed in an abscess-cavity containing a varying amount of foul pus. The walls of the cavity are formed by adjacent peritoneal surfaces, coils of intestines, cecum, and omentum bound together by adhesions.

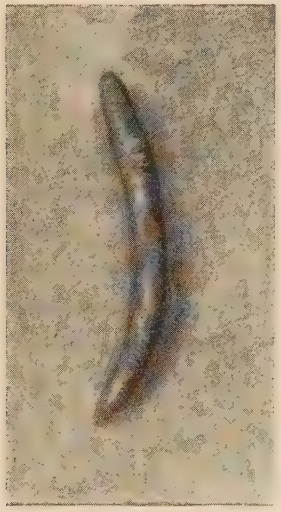

Fig. 271.—Symmetric involution of appendix (Morris).

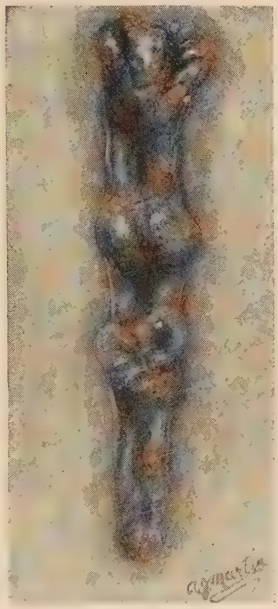

Fig. 272.—Nodular involution of appendix (Morris).

The appendix lying in the abscess-cavity is congested and swollen, and may or may not be perforated or present areas of gangrene. It may be adjacent to the cecum or intestines or reach into the pelvis to the bladder or tubes and ovaries. Perforation, when present, is generally near the free end of the appendix.

5. *Gangrenous Appendicitis*.—The appendix is congested, swollen, thick, and red, with gangrenous areas of greenish-black color and usually already perforated. It is marked by the *absence of protective peritoneal adhesions*.

There may be a fatal septic peritonitis before perforation of the appendix. Patches of fresh fibrin may be present with serous or bloody turbid serum in the peritoneal cavity or adhesions with pus.

The condition is an acute gangrene due to thrombosis and occlusion of the blood-vessels. If the case is more chronic, some plastic peritonitis may be present.

A purulent appendix, an acute diffuse appendicitis, or a concretion may ulcerate through, so that any of these types—including the gangrenous—may be classified as *perforative appendicitis.*

6. *Protective Appendicitis or Harmful Involution of the Appendix* (Morris).—The vermiform appendix normally undergoes an involution process with replacement of the lymphoid, mucous, and submucous coat by connective tissue. R. T. Morris[1] notes that the nerve-filaments persist longer than other structures, and contraction of the connective tissue in some cases irritates these nerve-filaments, so that irritation of the ganglia of the bowel (Auerbach's and Meissner's plexuses) ensues and causes disturbances in the nearby intestines. This condition he characterizes as " fibroid degeneration of the appendix." [2]

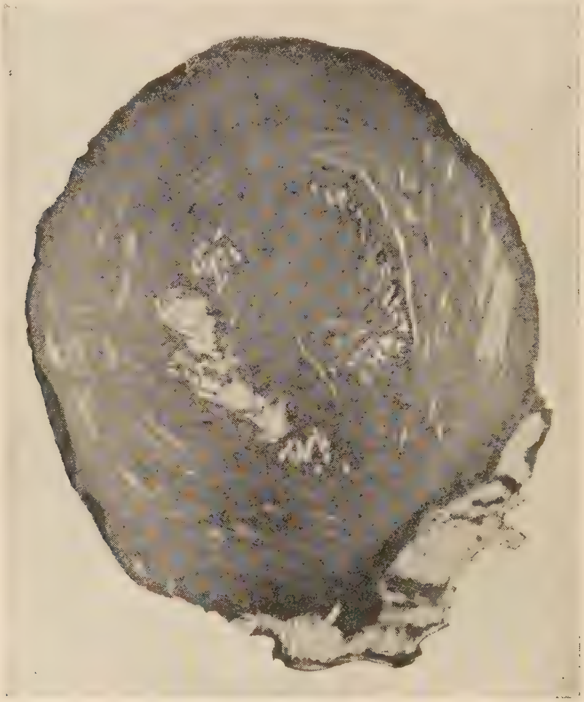

Fig. 273.—Transverse section of the appendix, showing replacement of the inner coats by connective tissue (Morris).

The appendix is at no time the seat of acute or chronic infection, and the condition is characterized by intestinal dyspepsia and other definite symptoms which will be described later. The involution may be symmetric or nodular (Figs. 271–273), and occurs most markedly toward the distal extremity.

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[1] Med. Rec., April 6, 1907; ibid., Jan. 8, 1910.
[2] Amer. Jour. of Surg., October, 1909.

R. T. Morris[1] classifies four kinds of appendicitis.

1. *Protective Appendicitis.*—An irritative lesion without infection.

2. *Appendicitis with Intrinsic Infection.*

3. *Syncongestive Appendicitis.*—An irritative lesion, due to serous infiltrates in the tissues of the appendix and occurring synchronously with similar congestion of neighboring tissues. For example, it may occur with obstruction of the lymph and blood circulation through certain diseases of vital organs and with loose right kidney.

4. *Appendicitis with Extrinsic Infection.*—An infective lesion due to bacterial approach from other structures lying outside of the appendix, such as from inflammation of the uterine adnexa.

A. V. Mosckowitz suggests a classification[2] which he has elaborated after a study of nearly 4000 cases at the Mt. Sinai Hospital. It embraces both the pathologic and clinical aspects.

I. Catarrhal inflammations.
  (*a*) Acute catarrhal appendicitis.
  (*b*) Chronic catarrhal appendicitis.
  (*c*) Acute catarrhal appendicitis with abscess.
  (*d*) Appendicular abscess.
II. Destructive inflammations.
  (*a*) Acute gangrenous appendicitis.
  (*b*) Acute gangrenous appendicitis with abscess.
  (*c*) Acute gangrenous perforative appendicitis.
  (*d*) Acute gangrenous perforative appendicitis with abscess.
  (*e*) Acute gangrenous or perforative appendicitis with gangrene of cecum.
III. Retention cysts.
  (*a*) Hydrops of appendix (mucoid contents).
  (*b*) Empyema of appendix.
  (*c*) Empyema of gangrenous appendix.
IV. In combination with blood infections.
  (*a*) Acute gangrenous or perforative appendicitis with thrombosis of mesentery.
  (*b*) Acute gangrenous or perforative appendicitis with portal pylephlebitis.
V. In combination with peritonitis.
  (*a*) Dry or fibrinous peritonitis.
  (*b*) Serous peritonitis.
  (*c*) Seropurulent peritonitis.
  (*d*) Purulent peritonitis.
  (*e*) Appendicular abscess with purulent peritonitis.

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[1] Protective Appendicitis, p. 97. Dawn of the Fourth Era in Surgery (R. T. Morris), W. B. Saunders Co., 1910.

[2] Amer. Jour. of Surg., Sept., 1910.

VI. Specific inflammations.
  (*a*) Tuberculosis.
  (*b*) Actinomycosis, etc.
VII. New growths.
  (*a*) Carcinoma.
  (*b*) Sarcoma.
  (*c*) Fibroma.
  (*d*) Diverticula, etc.

This table the writer believes of chief value as an index to the possible conditions that may be found at operation.

**Symptoms of Acute Appendicitis.**—The symptoms of acute appendicitis are modified by the character of the lesion, whether it is a simple catarrh, an appendicitis with the production of adhesions, a pus-cavity, or of the acute gangrenous type.

In general the cardinal symptoms of acute appendicitis are as follows:

1. Sudden pain in the abdomen in the right iliac region, or at times first epigastric or umbilical, which soon or gradually localizes in the right iliac fossa. Pains are continuous, increasing, or there are only exacerbations of pain. More rarely general abdominal pain is at first present.

2. Tenderness or pain on pressure in the right iliac region at McBurney's point. Often there is an area of resistance due to tumor or to muscular rigidity. Localized rigidity is frequently our main guide in children. The symptoms are often misleading in infants. Deaver[1] reports 300 cases.

Moreover, we must remember that in children the abdomens are small and their appendices are relatively long, so that sensitiveness at a distance from McBurney's point is more common than in adults.

3. Fever of moderate or severe type.

4. Gastro-intestinal disturbances may be present, such as nausea or vomiting, and frequently constipation.

5. In the *septic gangrenous type* I have, in a fairly large percentage of cases, observed a toxemic type of diarrhea with general abdominal pains at times, as the initial symptom, before localization of the appendix pain. This is evidently of septicemic character and has not, to my knowledge, heretofore been referred to as a symptom.

Rudolph Schmidt[2] assumes that the changes from normal in the intestinal flora of the feces, which occurs in appendicitis, may explain diarrhea in the early development of this disease; or possibly acute enteritis from dietary indiscretion may be the cause. The type of diarrhea with gangrenous appendix seems to the author *peculiarly septic* and is relieved by *appendectomy*.

6. In some cases the thighs and knees are flexed.

7. Irritability of the bladder, if the appendix extends down into the pelvis, may occur in some cases.

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[1] Jour. Amer. Med. Assoc., Dec. 2, 1910.

[2] Rudolph Schmidt, Pain.

*Appendicitis in Infants and Children.*—Appendicitis is more common in the male sex and usually runs a rapid and severe course. In a more recent article Deaver[1] states that "all cases of abdominal trouble in children are appendicitis until proved otherwise." There should be a systematic examination of all organs. Appendicitis in infants is difficult to diagnose, as the symptoms are often misleading. The younger the child, the more deeply the appendix lies in the pelvis. Urinary symptoms may predominate in these cases, for example, cloudy urine from edema of the bladder, tenesmus, and even retention. Pelvic abscess is common and hip-joint disease may be simulated. Rectal examination is of great importance as an aid to diagnosis, as an appendical abscess or enlarged appendix can frequently be determined by this method.

*Pain of Appendicitis.*—The pain may be sudden and violent, or at times intermittent and cramp-like, or even of a gnawing character or a dull ache. Sudden and violent pain in the initial stage does not by any means mean perforation, unless other symptoms are associated. In about one-half the cases the pain begins in the right iliac fossa; it may commence in the epigastrium, around the umbilicus, or even be diffuse, but gradually becomes localized within twelve to twenty-four or thirty-six hours, and usually sooner.

At times the pain is of a colicky type (the so-called appendicular colic, supposed by some to be due to constriction of the appendix in forcing out mucus through a lumen nearly occluded). Pain is increased on moving. It is often relieved by flexing the knees and thighs, especially the right thigh, and so relaxing the abdomen. This position is at times assumed by the patient.

*Palpation of the Appendix.*—Though some surgeons believe this to be an important procedure to render the diagnosis certain, Treves and Lockwood are very skeptical regarding the possibility of mapping out this organ. In the chronic cases palpation is of value, and the position and condition of the appendix can often be determined thereby.

In acute appendicitis the methods of forcible palpation recommended often necessitate the use of considerable pressure, and I believe the procedure highly dangerous. In the initial stages of acute appendicitis it is often impossible to at first determine the character of the attack, and traumatism in some cases can precipitate a rupture.

In the acute cases gentle palpation only should be used. Percussion, according to Rudolph Schmidt, will often demarcate the area of pain better than will palpation.

*Muscular Rigidity.*—There is usually rigidity of the right rectus muscle of varying intensity.

*Tenderness on Pressure.*—This occurs at McBurney's point and is of varying intensity, also at Morris' point, and Blumberg describes a new symptom. There are two points of great diagnostic value in appendicitis: tenderness at McBurney's point and at Morris' point (over the right lumbar ganglia).

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[1] Jour. Amer. Med. Assoc., Dec. 24, 1910.

*McBurney's Point.*—If a line be drawn from the anterosuperior spine of the right ileum to the umbilicus, a point 1½ inches from the spine along this is known as McBurney's point; and deep-seated tenderness on pressure over this point is diagnostic of appendical inflammation when taken in consideration with other symptoms (Fig. 274). Deep pressure also often causes reflex epigastric pain. Berthonier has recently pointed out that the examination in the left lateral position produces severe pain over McBurney's point in appendicitis, even in cases where this is not found with the patient in the dorsal position.

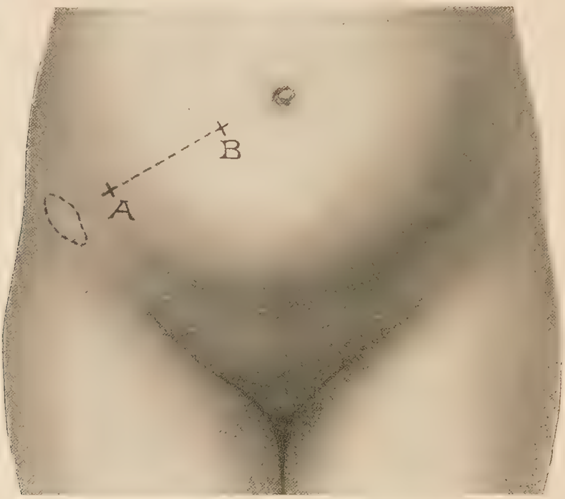

Fig. 274.—*A*, McBurney's point; *B*, R. T. Morris' point (lumbar ganglia) in appendicitis.

Mere superficial tenderness means irritation of the sensory nerves of the abdominal wall due to hysteria, etc.

Munro's point is slightly further out, where the same line crosses the outer border of the rectus.

*Lanz's Point.*—This observer draws a line between the two anterosuperior iliac spines, and divides this line into three equal parts. He claims that the first point of division corresponds to the base of the appendix and that McBurney's point is too high up.

*Morris' Point.*[1]—" Take another point on this same line, but 1½ inches from the navel, which lies over the right lumbar ganglia of the sympathetic system, and we have another point of diagnostic value when tenderness on pressure is located in this region.

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[1] Surgical Section, New York Academy of Medicine, Dec. 5, 1907. The author here quotes Morris' deductions.

"1. In the early *stages of an acute infective process* of the appendix, the right lumbar *ganglia are not tender.* (The left lumbar ganglia may be described for diagnostic purposes as lying 1½ inches to the left of the navel.) Under these circumstances the point here described is of *secondary importance,* while McBurney's point is of *prime consequence.*

"2. *A.* When an acute inflammatory process of the appendix has subsided, leaving a mucous inclusion or scar tissue, there may be no tenderness on pressure at McBurney's point, but there is tenderness *at the point here described* and no tenderness at the point of the left lumbar ganglia.

"*B.* When the appendix is undergoing an involution process, with replacement of its lymphoid *coats by connective tissue,* digestive disturbances and various local neuralgias may be due to irritation of nerve-filaments entrapped in the new connective tissue. There may be no tenderness at McBurney's point, but there is persistent tenderness at the point here described. There is no tenderness at the point of the left lumbar ganglia."

The above condition constitutes fibroid degeneration of the appendix.

"*C.* When the appendix is congested without the presence of infection, as in many cases of loose kidney, there may be little or no tenderness at the point here described. There is no tenderness at the point of the left lumbar ganglia."

The author wishes to state, as before, that this type of appendicidal congestion he believes *is not due to loose kidney pressure,* but is dependent on the *enteroptosis.*

"In irritations of pelvic origin, both right and left lumbar ganglia are tender. Take, for illustration, a case in which the appendix and the right Fallopian tube are bound together by adhesions. We are to decide whether certain symptoms proceed from the appendix or from the Fallopian tube. If the symptoms proceed from the appendix, the point here described is tender alone. If the symptoms proceed from the Fallopian tube, *both right and left lumbar ganglia* are tender together.

"To recapitulate: A patient comes in with the appendix in the form of a question mark. Right lumbar ganglia tender alone —appendix trouble. Right and left lumbar ganglia tender together —pelvic trouble. Neither right nor left lumbar ganglia tender— trouble somewhere cephalad from pelvis and appendix."

*Superficial Reflexes of the Abdominal Wall.*—In appendicitis the skin reflexes of this region are frequently absent or markedly diminished. The return of the lost reflexes is usually synchronous with other evidences of recovery.

Blumberg[1] describes a sign pointing to peritoneal irritation or inflammation, which he considers will be found of assistance in the diagnosis of all peritoneal conditions, and especially of appendicitis. It consists in the fact that on palpating the abdomen in the neighborhood of an area of inflamed peritoneum, not only is the downward pressure painful, but, if the examining hand is suddenly removed, the *abrupt recoil* of the abdominal wall also gives rise to pain. He has found that during an acute attack of appendicitis with peritoneal involvement, patients invariably state that the pain caused by the sudden removal of the hand is greater than that caused by the pressure, while if the inflammatory process is subsiding, the two painful sensations first become equal in intensity, and finally the pressure pain is greater than the other. He further believes that the sign is especially valuable in determining whether or not operation is indicated in early cases, since its presence indicates that the peritoneum has already begun to take part in the inflammation. Its sudden appearance is, therefore, a danger signal, while its gradual diminution in intensity points to a subsidence of the peritoneal reaction. The advantage of the method is that it does not require an absolute estimation of the degree of pain caused by the palpation, which is often difficult to obtain, but demands simply a comparison of the intensity of the two painful stimuli, which most patients are able to determine accurately.

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[1] Münchener med. Wochenschr., June 11, 1907.

*S. J. Meltzer's Method.*—Meltzer extends the right knee, at the same time flexing the right thigh, while making pressure over McBurney's point. This projects the psoas muscle against the appendix and causes deep-seated tenderness in the appendix to be more readily appreciated.

H. Illoway[1] holds that forced flexion and especially forced extension of the right thigh will cause pain in the appendix if inflammation is present.

*Rovsing-Chase Method.*—The patient is preferably placed on a hard low bed or table. The knees should be flexed and two pillows placed under the head and shoulders, giving a dorsal semirecumbent position, and thus rendering the abdomen flaccid for a satisfactory abdominal compression. The examiner stands on the patient's left, facing the feet. The palmar surfaces of the fingers of the right hand are placed at the lowest part of the patient's left inguinal region, and the fingers of the left hand are used to reinforce the right. Deep pressure is then made backward along the descending colon, the fingers being slowly drawn upward, toward and under the left costal arch. By this procedure, the lower portion of the descending colon is compressed and its gaseous content forced into the transverse, and thence into the ascending, colon. The pressure over the descending colon being maintained with the fingers of the left hand, the right hand is then removed and placed over the upper part of the descending colon, or, preferably, over the transverse colon, and the fingers are quickly and forcibly depressed. A gaseous *compression wave* will travel across the transverse and down the ascending colon, and on arriving at the cecum will produce distention, producing a typical sharp pain in the right iliac fossa, if inflammation of the cecum or appendix be present.

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[1] Archiv. of Diagnosis, July, 1908.

In many cases, especially in the acute catarrhal conditions or in the most virulent gangrenous type, no *induration* or swelling can be appreciated on palpation. Under such conditions forcible palpation in the endeavor to appreciate the appendix should be avoided.

*Percussion.*—In cases with adhesions, exudation without pus, or of abscess, a boggy or, rarely, fluctuating mass can be appreciated in the right iliac fossa, and percussion will give a dull area in the region of the cecum. Fecal accumulation as a result of constipation must be differentiated by the methods I have already indicated.

At times great irritability of the bladder is associated with this condition and the urine may be scanty and contain indican and albumin, and even acute nephritis may be present.

*Rectal* and *vaginal examination* often aid in localizing the condition if the appendix or abscess lie in the pelvis. In single women one should employ rectal examination. These methods also aid differential diagnosis. Inspection may occasionally show protrusion on the right side or the distention of general peritonitis.

If *tumor is present*, it may be variable in size, and it lies more frequently in the right iliac fossa. Its position, however, depends on that of the appendix, as already described.

*Temperature.*—An initial chill is rare. In the acute cases fever, even though slight, is present in the early stages. It may be only 99.5° F. or keep low, or even rise to 101° or 102° F., or to a considerable height.

Sometimes with circumscribed abscess there may for a time be only moderate temperature, and some cases of the virulent type will suddenly perforate, though the temperature be not high. In both of these the physical examination, the blood-count, and general symptoms will aid the diagnosis.

In general, a rise of temperature is significant of an active process, even though the temperature increase may be slight in degree and gradual in character. A slight increase in rapidity of the pulse is also suggestive of an acute process, and at times this is noticeably *much out of proportion to the temperature*, especially the *rapid pulse* in *gangrene* or *sudden perforation.*

**Gastro-intestinal Symptoms.**—Loss of the appetite and coated tongue are present. Emaciation may occur in cases of long duration.

In the severer cases vomiting is quite common. It may be one of the first symptoms occurring with the pain, and then cease, or it may continue for several days. On the other hand, it may come on later in the attack and denote an exacerbation of the inflammation. It consists of the stomach-contents, mucus, and bile; and in some cases it may be feculent. Associated with it there is at times hiccup.

Black vomit ("vomito negro appendiculaire") is the result of toxemia, producing hemorrhagic necrosis of the mucosa of the stomach and hematemesis.

A few cases have also been reported of intestinal hemorrhage associated with jaundice and albuminuria. These conditions are all evidences of a fatal sepsis. Acute ectasia or acute gastro-intestinal dilatation may also occur as complications.

*Bowels.*—In some cases the bowels are regular until the attack, when constipation ensues. In others there may be a previous history of constipation. Diarrhea of a toxemic type may be one of the initial symptoms in acute gangrenous appendicitis. It is probably of septicemic character. I have noted it in several such cases. Intestinal paresis may occur as a complication.

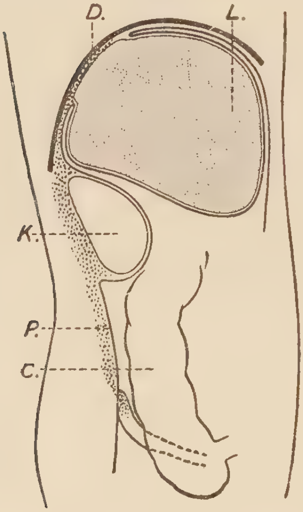

Fig. 275.—*D*, Diaphragm; *L*, liver; *K*, kidney; *P*, peritoneum; *C*, colon. The appendix lies behind the cecum and colon. The dotted area indicates the retroperitoneal spread of appendical inflammation to the cellular tissues around the kidney and the under surface of the liver (Cantlie).

*Tumefaction or Abscess.*—In cases of acute appendicitis in which adhesions are present, a tumor due to adhesions and exudation, or an actual abscess, the position of the mass is dependent upon the position of the appendix primarily and then upon the direction of the extension of the inflammation or burrowing of the pus. It may pass down into the pelvis and produce bladder and rectal symptoms, or those pointing to the tubes and ovary (right), and be palpable through the rectum and vagina. It may point below Poupart's ligament or simulate a psoas abscess. It may pass around in front of the cecum and superficial edema be noted in this region, or posteriorly to the cecum and cause pain in the flank or back, with swelling, and produce perinephritic abscess or lumbar abscess. It may pass inward to the left, even to the spleen, or produce obstruction through pressure or bands, or upward and produce subphrenic abscess (Fig. 275), and even perforate the diaphragm, pleura, and lungs. It may develop in a hernial sac.

At times the tumor appears at the lower border of the liver, when the tip of the appendix lies in this region. The condition must then be differentiated between retroverted appendical abscess and gallbladder or renal inflammation, such as infarction.

Brewer has shown that *tenderness at the costovertebral angle* is diagnostic of the latter; the urine analysis is also important, and especially differential analysis after catheterization of both ureters. The history will usually point to appendicular inflammation or disease of the gall-bladder, but operative procedure will alone settle some cases.

The value of Head's zones of cutaneous hyperalgesia as an aid to differential diagnosis will be referred to under that section.

The elements in the diagnosis of abscess formation are the gradual increase of the local tumor and the aggravation of the general symptoms. The abscess may perforate and cause a general peritonitis or the inflammation may extend to the peritoneum without perforation, so that there may be a slower process with various sacculated collections of pus.

The abscess may rupture through the skin or empty into the cecum, colon, small intestine, bladder, rectum, or pelvis of the kidney.

**Fulminating Type of Acute Appendicitis.**—This is the most dangerous and fatal of all. This type of appendicitis can be subdivided into two clinical varieties. Both are characterized by the rapidity of the pathologic changes in the appendix and by the absence of protective peritoneal adhesions, so that general peritoneal infection occurs quite rapidly.

In the first class the pain in the region of the appendix and some of the subjective and objective symptoms are acute and quite marked, but not all of them.

In the second class, and by far the most dangerous because frequently undiagnosed until the general infection has occurred, the patient complains of no marked subjective symptoms; in fact, may say he is quite comfortable, and the objective symptoms are not marked. There are peculiarities in the pulse, temperature, and especially in the blood changes which tell the story.

Acute gangrene of the appendix, with or without perforation, is the pathologic condition generally found in operation; though I have also seen an acute diffuse or purulent inflammation of the appendix, either with perforation or without it, produce the condition. In some cases perforation was apparently due to ulceration from a fecal concretion which was found in the cavity. Perforation undoubtedly is present in many cases, but I agree with Riegel that acute virulent infection of the peritoneum, commencing in the region of the appendix, will produce the same clinical symptoms.

*In the first class of cases* the patient may have given a history of previous attacks or have had some indefinite abdominal symptoms for several days, or may be attacked without warning, as in the middle of the night, with severe pain in the abdomen. It may not at first be referred to the appendical region, may be epigastric or umbilical pain, but gradually localizes there. There is no tumor, the muscles are rigid, the appendix tender. The abdomen rapidly distends. The patient is anxious and looks sick. The temperature at first may not be elevated much. The pulse is *rapid and out of proportion* to the temperature. General symptoms of peritonitis rapidly ensue. In others the pulse may be slow at first and a low temperature with local signs of appendicitis, but acute history, nausea, vomiting, and marked constipation. The pulse later becomes more rapid, high temperature, distention, coated tongue, and general tenderness.

In others the first symptoms are of collapse, with subnormal temperature, rapid pulse, cold and clammy skin, respiration increased in frequency, followed by symptoms of general peritonitis.

*In the second class of cases* the condition may be very deceptive. The patient, in perfect health, may suddenly complain of general abdominal pains. I have seen a number with *diarrhea* of toxic character as the initial symptom. The patient may impute the symptoms to dietary indiscretions. The tenderness may be diffuse or equal on both sides, and gradually it localizes in the appendix region. Even so, the tenderness at McBurney's point may not be very acute, and the rigidity of the right rectus not very marked or very slight. The patient may state that he feels quite comfortable. The temperature may be moderate (100° F.) and the pulse 100.

*The temperature tends to gradually creep up, the pulse to increase in rapidity out of proportion to the temperature increase,* which last may be slight, *and the character of the pulse changes;* the patient looks more sick, but still complains of no special symptoms except slight pain or exacerbations of it in the right side, and still feels comfortable.

The blood examination shows in many cases moderate leukocytosis (14,000 to 16,000), but marked increase in the polynuclears (88 to 92 per cent.). Hyperinosis is present. The patient presents as yet no symptoms of general peritonitis, but nevertheless the sepsis is marked in these cases, and unless immediate operation is performed will have a virulent type of septic peritonitis.

A patient of mine operated on by Hartley presented the mild type of symptoms just described: Diarrhea at 4 P. M.; no appendical pain or tenderness; localized appendical pain at midnight, when the surgeon was at once called in. Differential blood-count at 8 A. M. and 1 P. M., showing a gradual increase in polynuclears and low leukocytosis (15,000 to 16,000). The patient was quite comfortable; no distention; slight pain over appendix, but the temperature and pulse slowly creeping up.

At 5 P. M. operation at the New York Hospital; acute gangrene of appendix and commencing peritonitis; ultimate recovery.

*The examination of the blood is thus imperative* when possible. Brewer,[1] however, reports one case of acute gangrenous appendicitis in which the blood count was within the normal limit, a temperature of only 99.2° F., and the only evidence of severe disease was a well marked rigidity of the lower half of the right rectus muscle. One must also remember that in the course of tabes, acute appendicitis has occurred even with perforation, and with the formation and rupture of an abscess or with diffuse peritonitis, with little or no pain, and with very few local evidences of an inflammatory process. Connors[1] reports an interesting case. The patient had chills, irregular temperature, later vomiting and leukocytosis. There were no pain, tenderness, and muscular rigidity. Autopsy showed perforative appendicitis and diffuse peritonitis. There were found the spinal lesions of tabes dorsalis.

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[1] Masked Appendicitis, N. Y. State Jour. of Med., March, 1910.

**The Blood in Acute Appendicitis.**—Hyperinosis (increased fibrin in the blood) has been demonstrated by E. E. Smith and Bartlett[2] to be more marked in direct proportion to the involvement of the serous surface, and hence, *depending on its degree*, is suggestive of proportional peritonitic infection.

One of the *most important factors in the determination as to operative procedure in appendicitis and as to the relative severity of the case is the differential leukocyte count.* Charles Langdon Gibson has especially pointed out that it is the disproportion between the percentage of polynuclear cells and the total leukocytosis that is important.

The chart (Fig. 276) assumes that 10,000 leukocytes per cubic millimeter is the upper limit of ordinary normal leukocytosis, and that 75 is the normal percentage of polynuclears. Gibson further assumed that in inflammations which are well resisted the polynuclear cells are increased approximately by 1 per cent. for every 1000 leukocytes above the normal 10,000 per cubic millimeter. Then in the chart the *horizontal* line will indicate a leukocyte count of 11,000, with 76 per cent. of polynuclears, whereas the *rising* line represents a leukocytosis of 11,000, but with 86 per cent. of polynuclears.

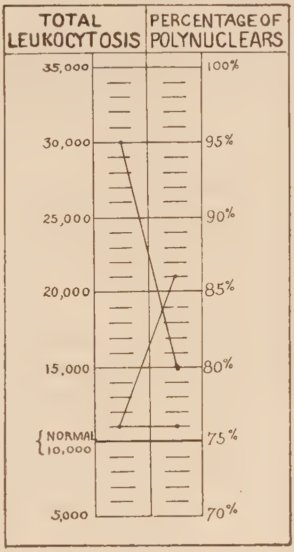

Fig. 276.—C. L. Gibson's differential chart for leukocytosis.

If the line connecting the total leukocytes and the percentage of polynuclears runs fairly *horizontal*, it indicates a lesion that, whether severe or not, *is well borne* and, therefore, of good prognosis.

If the line runs *upward* from the leukocyte to the polynuclear side, it indicates a rather severe lesion and less resistance.

Fatal cases all have a rising line.

A *falling* line (*e. g.*, leukocytosis of 30,000 with 80 per cent. of polynuclears) means a *mild lesion;* in appendicitis it would probably indicate an abscess well shut off, with little febrile or constitutional disturbance.

Gibson's conclusions are as follows: The differential blood-count and its relation to the total leukocytosis is the most valuable diagnostic and prognostic aid in acute surgical diseases that is furnished by any of the methods of blood examination.

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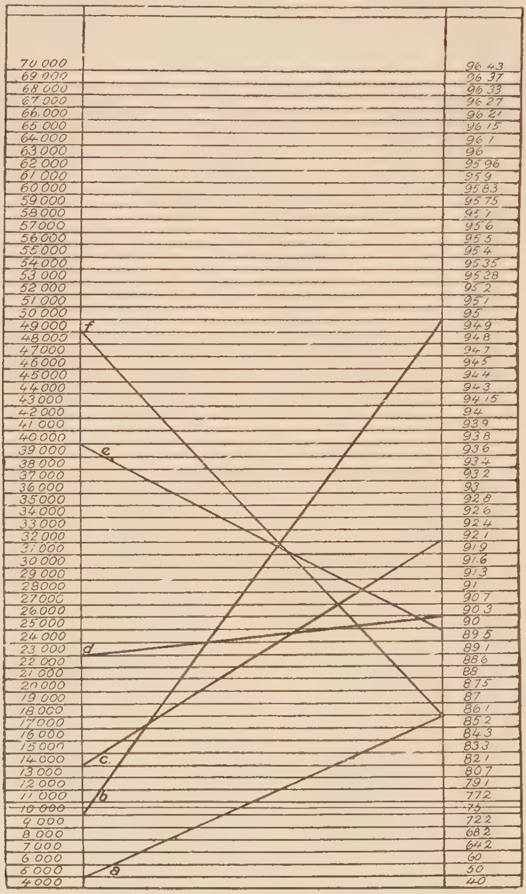
[1] Jour. Amer. Med. Assoc., Oct. 22, 1910.

[2] Blood Reactions of Inflammation, Med. Rec., Feb. 8, 1908.

It is of chief value in indicating fairly consistently the existence of suppuration or gangrene, as evidenced by an increase of the polynuclear cells disproportionately high as compared to the total leukocytosis.



*a.* Gangrene of the gallbladder. Leucocytes, 4,300; polynuclear, 85.5 per cent. Fatal.
*b.* Perforation, typhoid fever. Leucocytes, 10,000; polynuclear, 95 per cent. Fatal.
*c.* Puerpural sepsis. Leucocytes, 14,000; polynuclear, 92 per cent. Fatal.
*d.* Gangrenous perforative appendicitis. Leucocytes, 23,000; polynuclear, 90.2 per cent. Recovery.
*e.* Appendicitis, local peritonitis. Leucocytes, 40,000; polynuclear, 89.9 per cent. Recovery.
*f.* Appendicitis, spreading peritonitis. Leucocytes, 49,000; polynuclear, 85.6 per cent. Recovery.

Fig. 277.—Blood-count chart (Sondern).

The Gibson chart is to a certain extent schematic, as it is only applicable to a leukocyte count of about 25,000, since if it were 30,000 or over there could only be a rise of 5 units in the polynuclears, and this would be 100 per cent., which never occurs. It would also suggest a good prognosis according to his unit system of rise and fall. One can apply Gibson's principles for the higher counts by the chart (Fig. 277), as suggested by Coons and Bratton.[1] Sondern substantially employs this same type of chart.

E. E. Smith[2] shows that the differential blood-count is an indicator of the *activity of the process* and not *invariably of gangrene;* but if the absolute *leukocytosis is low* (below 15,000), with *high polynuclears*, it is *probably* gangrene.

Gibson further holds that the greater the disproportion the surer are the findings, and in extreme disproportions the method has proved itself practically infallible.

As the relative disproportion between the leukocytosis and the percentage of the polynuclear cells is of so much more value than the findings based on a leukocyte count alone, this latter method should be abandoned in favor of the newer and more reliable procedure.

The negative findings, showing no relative increase or even an actual decrease of the proportion of the polynuclears cells, while of less value, show with rare exceptions the absence of the severer forms of inflammation.

In its practical applications the method is of more frequent value in the interpretation of the severity of the lesions of appendicitis and their sequelæ.

Though N. E. Ditman, in a paper read before the Obstetrical Section of the Academy, November, 1906, criticizes the differential count as of doubtful value, Sondern[3] shows that in cases quoted the pus was encapsulated in such a way that no toxic absorption occurred, and also demonstrates that polynuclear increase may occur in other than suppurative conditions.

It is a well-known fact that such encapsulated abscesses may occur without marked constitutional symptoms, *but physical signs determine* their presence.

In this type the processes are less active, and this is probably the chief cause.

I have seen a very low leukocytosis with a general fatal peritonitis, the system evidently being overwhelmed by the poison. The physical signs, with increased *polynuclears* and *hyperinosis*, aid our diagnosis in all but exceptional cases.

A low leukocytosis with high polynuclear count also shows poor resisting power to the infection.

To recapitulate: We may have numerous types of acute appendicitis, which clinically are as follows:

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[1] Prognostic and Diagnostic Value of the Leukocytes and Differential Count in Acute Abdominal Infection, N. Y. Med. Jour., July 21, 1909.

[2] Blood Reactions of Inflammation, Med. Rec., Feb. 8, 1908.

[3] N. Y. Med. Jour., June 26, 1907.

1. Simple catarrhal appendicitis with mild symptoms, lasting a few days to a week or ten days; often secondary to colitis, intestinal disturbances, or fecal impaction in the caput coli; blood changes are moderate and the attack soon subsides under medical treatment combined with the ice-bag. It may never recur.

2. Acute appendicitis (diffuse), symptoms more severe; may be adhesions or slight exudation; temperature higher; more marked tenderness; tumor often palpable, but may disappear. It may become chronic or recurrent, or occasionally perforate.

3. Acute appendicitis, with abscess, chills, tumor, etc., present. Abscess may perforate.

4. Fulminating type, perforation or gangrene.

**Remote Effects of Acute Appendicitis.**—They are as follows: Hemorrhage from perforation of a blood-vessel; suppurative pylephlebitis; pyelitis; thrombosis of the iliac or femoral veins; pulmonary embolism; strangulation of the bowel; subsequent symptoms may occur due to incomplete removal or subsequent adhesions. Hartley has noted intestinal paresis in mild cases, with symptoms of ileus resulting; also infection of the mesenteric glands, subsiding in some cases after appendectomy, and in others causing subsequent inflammation. Cellulitis of the abdominal wall has occurred.

Postoperative stomach and intestinal hemorrhage after appendicitis operations are described by Schwalbach,[1] who collected 30 cases. They are more frequent in children and in men. There are hemorrhages, erosions, and ulcerations in the stomach and intestines due to thrombosis in the circulatory system of the omentum and mesentery.

**Chronic Appendicitis.**—This may follow an ordinary acute attack or be chronic from its incipiency.

The latter often is secondary to chronic intestinal catarrh. The patient has frequently a continuous feeling of discomfort in the appendical region; slight tenderness on pressure; generally intestinal and frequently nervous disturbances associated, also obstinate constipation or gastric disturbances. Some of the symptoms may simulate gastric or duodenal ulcer, or cholelithiasis or achlorhydria hæmorrhagica gastrica may be secondary, or there may be persistent vomiting. There may be hyperacidity or hypo-acidity of the stomach, or achlorhydria or the findings may be normal. Renal disease or renal calculus or ureteral calculus may be simulated. In my own experience vomiting is not so prominent a symptom in chronic appendicitis as with gastric ulcer, and when vomiting occurs it more frequently consists of food rather than sour liquid. Nausea is more characteristic. There may be pain or, more frequently, *distress* shortly after eating, which may continue for a time. The pain may be epigastric, but is more frequently indefinitely or plainly abdominal in chronic appendicitis. Pylorospasm may occur. Orthoform or anesthesin relieves the pain of ulcer, but not that of chronic appendicitis. Slight tenderness or discomfort on pressure at McBurney's point may be present. This may disappear for periods. At times no tenderness can be elicited at McBurney's point, but is noted at Morris' point. There may be exacerbations of acute attacks.

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[1] Deutsch. Zeit. für Chir., Bd. 95, H. 1–5, p. 141.

Indiscretions in diet often cause exacerbations of the symptoms. In some of these cases there is simply a chronic catarrhal condition, but more frequently angulation with adhesions.

Serious conditions are at times found in the appendix in these chronic cases, and a brief reference to such is important. A patient, F. J. G., age thirty-five, financier, with a history of a mild appendical attack three years ago, presented the following symptoms: For a period of three weeks a feeling of general malaise, appetite capricious, pains in the neck and limbs, occasional belching, considerable intestinal gas; occasional discomfort and tenderness on pressure in the right iliac region. Patient at times awakened by gas-pressure; occasional chilly feelings. He came to the office for examination; temperature 99° F.; slight tenderness on pressure over the appendix; somewhat higher and nearer median line than usual; leukocytosis 10,000; polynuclears normal. At end of nine days, on subsidence of symptoms, appendectomy by Frank Hartley. The base of the appendix and about 2 inches of the body was firmly bound to the cecum, lying almost vertical. There was then an acute bend (right angle), and about 1½ inches pointed out free into the abdominal cavity toward the median line. The appendix lay high up. The tip was bulbous, contained two small concretions, and there was a small superficial ulceration. With an acute attack, perforation and general peritonitis would have resulted.

Chronic appendicitis is the type where palpation by Edebohl's method is safe and of value, and where at times the enlarged appendix can be appreciated.

The patient lies on his back with the thighs flexed, and the examiner, placing three or four fingers of the right hand, palm side downward, draws them over the abdomen from the umbilicus to the anterosuperior spine of the ileum, exerting considerable pressure. The appendix can be recognized as a firm cord. Morris reinforces this hand with the three fingers of the left hand. Gentle palpation by the following method is often preferable (Morris): First seek the ascending colon as a landmark. It is palpated by pressing the three fingers of the left hand gently beneath the right rectus muscle on a plane above the navel, and then drawing the fingers away so that the colon slips out from beneath them. Second, follow this landmark down until the cecum is reached. Gently indent the abdominal wall with these fingers at the site of the appendix. With the other hand on the other side of the abdomen, press back and forth, so the cecum is carried back and forth beneath the fingers. If this fails, press the feeling fingers beneath the inner edge of the cecum, so the latter will be lifted over their tips and make lateral pressure on the opposite side as before.

It has been at times recommended to examine with the patient standing erect or bending slightly forward, but the dorsal posture is preferable.

In the chronic cases there is generally no temperature unless subacute or acute exacerbations occur.

**Protective Appendicitis or Harmful Involution of the Appendix.** — Symptoms of harmful involution of the appendix (Morris):

1. Symptoms of auto-intoxication with attacks of headache, nervousness, poor appetite, etc. 2. Chronic intestinal dyspepsia. 3. Discomfort in the appendical region. 4. An appendix feeling hard and narrow on palpation. 5. Hyperesthesia of the right lumbar plexus.

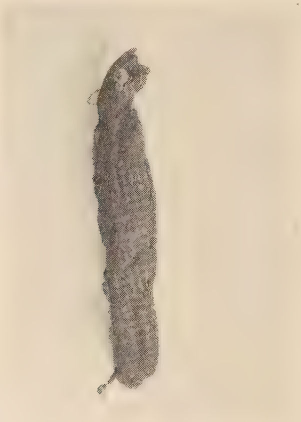

Fig. 278.—An appendix which was quite normal in external appearance, but it felt harder than normal on palpation, and longitudinal section shows the inner coats to have been replaced by connective tissue, with total obliteration of the lumen. The patient had suffered from occasional attacks of pain in the appendical region, and from intestinal dyspepsia, both of which disappeared on removal of the appendix (Morris).

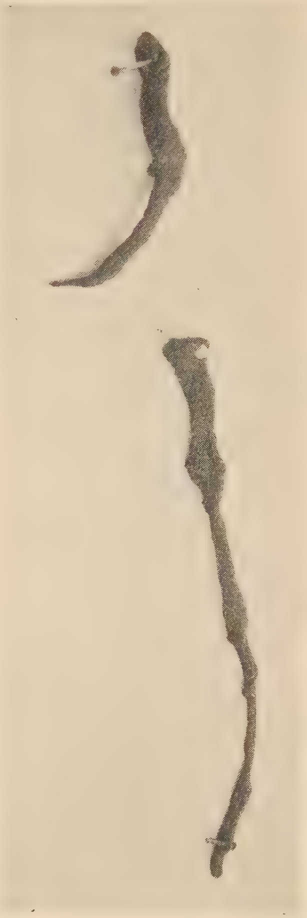

Fig. 279.—Two appendices in which all structures had practically been replaced by connective tissue, but enough nerve-filaments had remained to cause persistent intestinal dyspepsia, for which the patients had received treatment by authorities without permanent result, until the appendices were removed. Both patients then became well (Morris).

There are persistent distention of the cecum and ascending colon, with gas and a sensation of discomfort in the appendical region. In some cases there may be acute local pain and tenderness, but not enough to send the patient to bed. They are rather apt to stay up and about on account of their general irritability. In others there is merely a sensation of warmth in the appendical region, which fluctuates in intensity from day to day and may be sometimes absent. In Figs. 278, 279 are depicted three appendices which were removed for this condition.[1] The patient has a tendency to press upon the abdomen at that point or to lean against a table. The sensation may pass away for a few hours or days, but tends to recur and last for years.

On palpation the involution appendix feels narrow and hard. There is no history of acute or chronic appendicitis. The type generally occurs in those over twenty-five or thirty years of age.

**Diagnosis and Differential Diagnosis.**—Abdominal pain, becoming localized in the right iliac fossa; tenderness at McBurney's point; rigidity of the right rectus; temperature; rapid pulse; gastro-intestinal disturbances; in some cases the presence of tumefaction and in others the subsequent development of peritonitis—are all diagnostic of appendicitis. In addition, there are the differential leukocyte count and hyperinosis, which are an aid to diagnosis.

Renal colic with calculus impacted in the right ureter may simulate appendicitis, but there are the history of acute pain in the kidney, burning sensation of the urine, and drawing up of the right testicle, with sand, gravel, or blood, etc., in the urine. The *x*-ray may give information. Movable cecum[2] has been mistaken for appendicitis.

With Dietls' crisis there are the movable kidney, the history of the attack, and the kidney is swollen and sensitive.

In intestinal colic the pain is relieved after passage of flatus.

In biliary colic the pain radiates to the back and up to the right shoulder, usually with a previous history of gall-stones, etc.

In perforation of the gall-bladder or duodenum the contents gravitate toward the appendix; and this possibility must always be considered in apparently acute perforative appendicitis. In these cases the sudden acute pain occurs in the epigastrium and right hypochondrium. Osler, in the Johns Hopkins Bulletin, July–August, 1904, in an article entitled "The Surgical Importance of the Visceral Crises in the Erythema Group," calls attention to the fact that abdominal pain in the erythema group occurs in angioneurotic edema; with simple urticaria; with Henoch's purpura; with erythema multiforme, and in other conditions in which skin lesions only occur late in the disease. Musser, in Amer. Med., March, 1904, reported cases. Osler describes purpura, various types of erythema, urticaria, and angioneurotic edema with visceral lesions in his Practice of Medicine. A. B. Johnson[1] reports cases of Henoch's purpura with initial abdominal symptoms, colic, pain, rigidity, vomiting, temperature, leukocytosis, etc., the cases closely mimicking *acute appendicitis*. In fact, until the eruption and joint lesions appeared, diagnosis was impossible. In women the differential diagnosis between an inflamed low appendix and salpingitis is sometimes difficult, as they frequently are associated.

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[1] Protective Appendicitis, pp. 99–103. Dawn of the Fourth Era in Surgery (Morris).

[2] With movable cecum there is local distention (tympanites) of the cecum, and the condition is usually associated with enteroptosis and its symptoms. Symptoms are not acute.

Tenderness of both lumbar ganglia, however, shows pelvic[2] trouble, and when the hypersensitiveness is greater on the right side, that the appendix and tubes are both diseased; with salpingitis, there is no tenderness at McBurney's point with the patient on the left side.

With pneumonia, especially central near the right base, and with diaphragmatic pleurisy, there is occasionally in the early stages pain *transferred to the right iliac fossa*, and mistaken for appendicitis. This possibility must be considered. The physical examination of the lungs and pulmonary symptoms should be carefully observed. Determination of Head's zones is of value; the zone for the lungs is from the first to the ninth dorsal segment, chiefly the third, fourth, and fifth, which give hyperalgesia over the thorax. With appendicitis hyperalgesia is below the umbilicus.

Hyperesthesia of the right lumbar ganglia is *absent* in pneumonia without *involvement of the appendix*. In other words, no hypersensitiveness of the right lumbar ganglia on deep pressure, then no appendicitis. Look for inflammation elsewhere according to Morris.

In typhoid fever there may be pain in the right iliac fossa and appendicitis is often a complication.

In simple typhoid there is no leukocytosis, but leukopenia.

In typhoid with appendicitis, in addition to the right iliac pain and tenderness, the presence of leukocytosis, increased polynuclears, and hyperinosis make the diagnosis of appendicitis. Luckett[3] has reported a case of torsion of the greater omentum complicated by acute appendicitis, in which the diagnosis of acute appendicitis with probable abscess was apparently justified by the symptoms. This is an interesting possibility to remember.

As an aid to differential diagnosis in affections of the viscera, the determination of Head's zones (cutaneous hyperalgesia) is of value. This is especially true, I believe, in the differentiation of appendicitis from the conditions to which I have just referred. Head found that in many visceral affections, if the sensitiveness of the skin was tested by running a pin point over the cutaneous surface, there could be shown to exist areas over which there was a more or less hypersensitiveness to pain. These areas were constant and distinct, could be mapped out on the surface of the skin, and, when present, were almost an infallible sign of an affection of the organ to which they corresponded. The skin tenderness was superficial and extended over definite areas which never overlapped one another. Each area or zone of hyperalgesia had a " maximum region" which often corresponded to the seat of pain. These areas were sensitive to heat and cold, but not to simple touch.

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[1] Conditions Simulating Appendicitis, N. Y. State Jour. of Med., March, 1910.

[2] The New Point in Diagnosis Between Appendicitis and Tubal Disease (Morris), Amer. Jour. of Obstet., 1909, vol. lx, No. 5.

[3] Jour. Amer. Med. Assoc., April 23, 1910.

The areas *corresponded to segments of the spinal cord*, not to the distribution of peripheral nerves or spinal nerve-roots. The zones were named according to the segments of the cord: cervical, 1 to 7; dorsal, 1 to 12; lumbar, 1 to 5; sacral, 1 to 4. They were broader in front at the median line, narrowed at the side of the body, and again broaden out near the spinal column. The zones, as a rule, never extend beyond the median line in front or behind. Head gives the following zones for the abdominal viscera.

Stomach, sixth, seventh, eighth, and ninth dorsal. Cardiac end, sixth and seventh dorsal, right. Pyloric end, eighth and ninth dorsal, left.

Liver, eighth, ninth, and tenth dorsal, right.

Gall-bladder, eighth and ninth dorsal, right.

Intestines, ninth, tenth, eleventh, and twelfth dorsal.

Colon, ninth, tenth, and eleventh dorsal.

Cecum and appendix vermiformis, tenth and eleventh dorsal, right.

Kidney, tenth dorsal, sometimes eleventh dorsal.

Ureter, eleventh and twelfth dorsal, first lumbar.

Bladder (first?), second, third, and fourth sacral.

Uterus, tenth, eleventh, and twelfth dorsal, first lumbar.

Appendages, eleventh and twelfth dorsal, first lumbar.

Head first tested sensitiveness to pain by pinching up folds of skin and later by stroking the skin with the point of a sharp pin. Elsberg[1] and Neuhof suggest the following method of examination:

" A sharp pin is held between the thumb and index-finger of the right hand, the nail of the index-finger resting on the patient's skin. The pin is then made to traverse slowly the surface of the skin, care being taken that the nail of the index-finger presses equally along the area examined. The patient is instructed to say ' now ' as soon as the pin stroke becomes painful.

" In examining the skin of the abdomen for hyperalgesic areas, the pin traverses the abdomen from side to side and from above downward; the points at which the patient complains of pain are marked. In this manner it is possible to map out areas on the skin, and when such an area has been found, the pin is made to approach it from all sides, so that its form and position can be determined. Care must be taken that the pressure of the pin point remains constantly the same, especially as the pin passes over the groin and slips off the costal border or over the crest of the ileum.

" After the zone has been thus mapped out on the skin the procedure is repeated a second time, and now it is a good plan for the operator to control both patient and himself by keeping both the patient's and his own eyes away from the pin.

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[1] Amer. Jour. Med. Sci., Nov., 1908.

" The hyperalgesia is sometimes so marked that the patient will shrink or cry out as soon as the border of the zone is reached. In very young children the examination is useless, but older children will give correct answers.

" If the examination is carried out in the manner above described it will be possible, in a large number of patients with visceral affections, to map out areas of hyperalgesia extending from the median line in front to the spines behind. The 'maximum' areas can often be mapped out lying within the boundaries of the zones; sometimes only the 'maxima' are present. Sometimes several 'maxima' are found in one zone."

The zones appear early in the course of visceral affections, and frequently persist throughout. They have been reported as appearing very early; for example, in the commencement of acute appendicitis, while the pain was still in the epigastric region and there was no local tenderness at McBurney's point, the zone for the appendix was discovered. Shortly after the typic symptoms appeared. One must remember the following (Elsberg):

1. The characteristic zone may appear after palpation of the diseased organ.

2. The hyperalgesic zone will not appear on examination until fifteen to thirty minutes have elapsed after removal of the ice-bag or hot-water bag, if such have been applied.

3. The disappearance of the zone, as a rule, follows relief of the lesion of the affected viscus.

4. The zones may disappear temporarily after repeated examinations in close succession. Later they reappear.

5. The disappearance of the zone, together with persisting or increasing symptoms, is probably a sign of ill omen.

6. The zones are not invariably present. While the absence of a characteristic zone in a suspected affection of an abdominal organ *does not mean* that there may not be disease of that organ, the *presence* of the *zone* means that there *is an undoubted lesion.* From this one must *not* conclude that the viscus which gives the zone is the one which causes *all the symptoms*, for we may get a zone from an organ which is secondarily affected.

7. The presence of areas of skin hyperalgesia *corresponding to several* viscera may mean a *combined lesion of several* adjoining *viscera*, although it may occasionally *mean disease* of the *spinal cord itself.*

8. The presence of a Head zone alone must not be the only factor in arriving at a diagnosis, but it must be used in conjunction with other signs and symptoms. When one is in doubt as to which of several viscera is the seat of the lesion, the presence of the charac-

teristic zone has been an aid; for example, in differential diagnosis between appendicitis (with retroverted appendix) and kidney and gall-bladder disease, or between appendicitis and salpingitis. It seems preferable to adopt Elsberg's method and speak of the zones by the names of the viscera to which they belong. The position of his zones vary a trifle from Head's, and are as follows:

Thus, the stomach zone corresponds to the seventh, eighth, and ninth segments of Head (according to Head, sixth, seventh, eighth, and ninth); the gall-bladder zone, to the eighth and ninth segments on the right side (same as Head's diagrams); the appendix zone, to the tenth and eleventh segments of Head on the right side. It will be found, in the description of the zones, that the limit of the posterior portions is not absolute. Thus, in describing the gastric zone, that it extends from the sixth to the tenth vertebræ approximately. The zones, except the gastric zone, stop sharply at the posterior median line, but their upper and lower margins are more variable.

*The zone appears on that side of the body on which the affected organ has its nervous connections, the side on which the organ is normally situated. If an organ belongs on the left side, the hyperalgesic zone will be found on that side, even if the organ, through disease or mobility, lies on the other side of the body.*

Those areas are called " objective zones " when the patient suffers actual pain as the stroking pin enters them. All less painful zones will be called " subjective zones." By an " anterior zone " we mean an anterior maximal area; by a " posterior zone," a posterior maximal area.

**The Stomach.**—The complete gastric zone was found to be uncommon. It extended as a broad belt all around the body. At times only a portion of it showed on examination.

In the median line in front it extends from the xiphoid almost to the navel; it then passes upward and backward on both sides toward the spine, where it extends from the sixth to the tenth vertebra (approximately). Incomplete zones are more frequent, either an anterior portion extending to the right or to the left, or on both sides of the anterior median line.

In Figs. 280 and 281, the various zones are depicted on the anterior and posterior surfaces of the body.

**The Duodenum.**—The duodenal zone lies between the gall-bladder and the appendix zones. It lies almost completely to the right, but occasionally extends slightly to the left of the anterior median line. Anteriorly it is broad; its upper limit is about on a horizontal line midway between the umbilicus and the ensiform cartilage; its lower border is a little below the umbilicus. It extends backward and slightly upward, and narrows; at the anterior axillary line it is very narrow (about 1½ inches); it then becomes broader, and is lost about the midscapular line. It corresponded roughly to the ninth dorsal zone of Head.

With a *perforating duodenal* ulcer, intestinal *contents gravitate* to the *appendical region.* If the ulcer was occult, differential diagnosis from appendicitis may be difficult. *Presence of the typic zone may prove of assistance.*

**Gall-bladder and Liver.**—This zone is present in acute affections of the gall-bladder more often than in any other acute intra-abdominal affection. In these cases the Head zone has often been a valuable diagnostic aid. In many cases an enlarged, tender, and palpable gall-bladder makes the diagnosis easy, but the recognition of the disease is often difficult or impossible in stout patients without jaundice, with marked abdominal distention and rigidity. These patients may refer their pain to the right lower abdomen, and may have their tenderness in this region. Acute intestinal obstruction, acute pancreatitis, or acute appendicitis are diagnoses often made. In some patients the presence of a zone of hyperalgesia has been the only localizing sign.

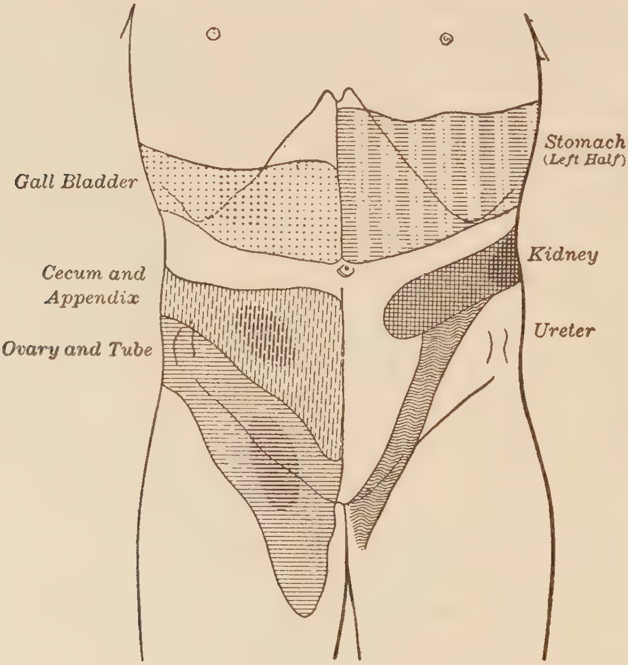

Fig. 280.—Head's zones. The general location and outline of the zones of cutaneous hyperalgesia for some of the abdominal viscera. Anterior view. The maxima are deeply shaded. Only the left half of the gastric zone is given. The ureteral zone consists of a series of maxima (diagrammatic) (Elsberg and Neuhof).

The gall-bladder zone lies in the right half of the abdomen, above the level of the umbilicus. The complete zone starts exactly at the median line in front, extending from some distance below the xiphoid to a short distance above the navel. Tracing it backward, it slants obliquely upward and becomes narrow, passing partly over and partly below the costal arch. It is narrowest at the midaxillary line, where it is about 2 inches wide. Posteriorly it becomes broader, and at the spines it is about as wide as in front. In some cases more or less of the anterior portion only has been present (maximal area).

**Kidney and Ureter.**—The kidney zone is wide at the posterior median line, where it begins, and gradually narrows anteriorly. Its greatest breadth is at the spinal column. It narrows to make a triangular area, with a rounded apex, situated a little to that side of the anterior median line on which the zone lies. It never quite reaches the anterior median line. Each zone is strictly limited to its half of the body. There is no difference in contour between the right and left kidney zones. The kidney zones are complicated by the additional ureteral zones that are present in certain cases. The ureteral zone springs, so to speak, from the lower margin of the kidney zone at the anterior axillary line. In an average adult it is about 3 inches wide at its beginning. It narrows in its downward course, and passing obliquely downward and forward, it terminates on its side of the penis and scrotum in the male; the labia in the female. After the first narrowing it widens again well below the umbilical level.

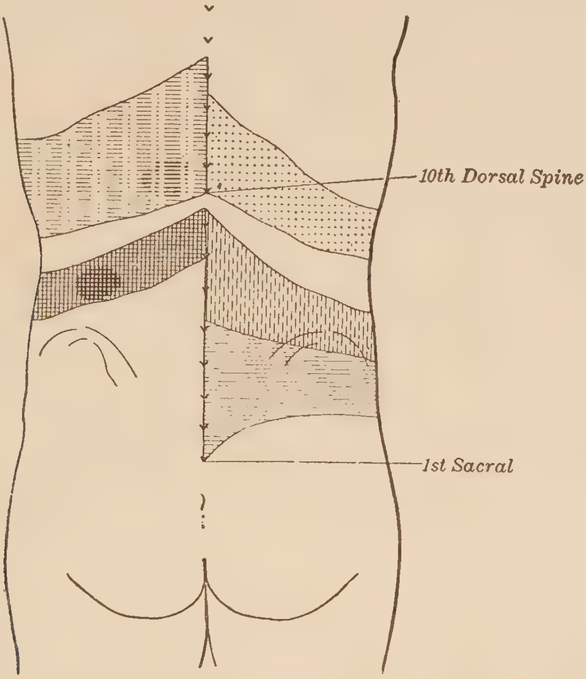

Fig. 281.—Head's zones. The general location and outline of the posterior parts of the zones (diagrammatic) (Elsberg and Neuhof).

In the male, it can be ascertained that the zone spreads fan shape to the anterior median line over the pubic area and its half of the scrotal and penile skin. There are anterior and posterior kidney maximal areas. The ureteral zone seems to be made up of a series of maxima. The kidney and ureteral zone is most often present, as in the other intra-abdominal affections, in the presence of pain and tenderness.

In the differential diagnosis of septic infarction of the kidney, pyelonephritis, etc., from retroverted appendix (appendicitis), determination of the kidney zone is of value, taken in connection with Brewer's sign, urinary analysis, etc. (author).

**Vermiform Appendix.**—The zone begins at the median line in front, sometimes a little to its left, from a point a short distance below the umbilicus to one equally distant from the symphysis pubis. It narrows toward the anterior axillary line to a width of about 2 inches (average adult). From this line it widens and spreads to the posterior median line from the eleventh dorsal to the second lumbar spines (approximately). At the anterior median line there is often a tongue-like downward extension of the zone (Figs. 280 and 281). There is an anterior maximal area which is sometimes present alone. It may be that the "appendix" zone is really an "appendix and cecum" zone, because the cecum is so frequently involved in appendicitis. Sometimes, when an ice-bag has been employed over the appendix region, only the posterior half of the zone is present.

Diagnosis has been aided in a considerable number of the patients by the presence of the zone, especially in that large class of acute cases in which the abdomen is rigid and there is no palpable mass. The zone has been of the greatest value in helping to differentiate between diseases of the appendix and those of the gall-bladder or right uterine adnexa.

The absence of a zone is of no significance. If a patient complains of symptoms which resemble appendicitis, and a zone is not present in the right lower abdomen, it is well to look elsewhere for hyperalgesia. Thus in cases of *beginning pneumonia* that had considerable *pain*, *tenderness*, and *rigidity in the right iliac region*, the presence of *hyperalgesia over the thorax first lead to careful examination of the lungs*.

**Intestines.**—Head gives the zone as corresponding to the ninth, tenth, eleventh, and twelfth dorsal segments of the spine. Elsberg does not find these uniform. He shows, however, uniform zones in ileocecal tuberculosis and in perforation of the ileum.

**Ileocecal Tuberculosis.**—There is a large area of hyperalgesia occupying the whole right lower abdomen down to Poupart's ligament, often extending a little to the left of the median line, and posteriorly becoming lost about the posterior axillary line.

**Perforation of the Ileum.**—The zone resembles that of ileocecal tuberculosis, but extends more to the left of the median line.

**Uterus and Adnexa.**—Head describes differences between the zones for the uterus, the ovary, and the tube. Elsberg differs somewhat: The zone for the right adnexa lies on the right half of the median line; that of the left adnexa on the left half; the zone for the uterus is a combination of the two. There is no difference between the zones for the right and left adnexa. Beginning some distance above Poupart's ligament, the upper margin of the zone runs parallel to it, and pursues this obliquely upward course to the spine of the second lumbar vertebra (approximately). The lower margin is a long, tongue-like process that extends half-way down the thigh on its inner aspect. The lower margin, as it passes a short distance below the anterior superior spine of the ileum, approaches the upper, the average breadth of the zone here being 3 inches. The lower border then passes horizontally backward over the buttock to reach the posterior median line partly over the sacrum. Sometimes the upper half of this zone is better developed, sometimes the lower; these may be considered maxima.

Diagnosis in the diseases of the uterus has not been aided by the presence of a zone. Elsberg states that in about half of the cases of dysmenorrhea and of endometritis with pain the zone was present. Some of the cases of retroflexion, retroversion, anteflexion, and prolapse showed the zone. It was present in the 5 cases of uterine polyp that he observed (all of them had pain). It was not present in tumors of the uterus, except in a few cases.

In diseases of the tubes and ovaries, especially those of the right side, the zones have been of diagnostic value.

I can substantiate these observations of Elsberg and Neuhof, and believe that the tests[1] for Head's zones are a valuable aid in differential diagnosis, especially in appendicitis.

**Prognosis.**—There is always an element of *uncertainty in every case of appendicitis*, and it is well to be guarded in every acute case when expressing an opinion; for an apparently simple case may suddenly show dangerous or even fatal symptoms. It is a well-known fact that in the simple catarrhal cases there may never be but one attack, perfect recovery resulting.

Many cases of exudation or abscess, especially of the old cases of so-called perityphlitis, have recovered without operation.

Sahli has collected 7213 cases; of these, 473 were operated on, 6740 were not.

Of the latter, 591 (8.8 per cent.) died; 6194 (91.2 per cent.) recovered; recurrences took place in 4593 cases; of these 3653 recovered without a second recurrence.

Nothnagel claims that 80 per cent. of cases of circumscribed appendicitis recover under medical treatment.

The fact that a patient has recovered under medical treatment from one or two attacks during a period of several years is no guarantee that a fatal issue may not ultimately occur. If a simple catarrhal attack occurs, with moderate symptoms and no marked changes in the blood, the prognosis for immediate recovery is certainly favorable. If no attacks occur during several years, the chances of subsequent attacks are lessened. In all statistics of a second attack and subsequent apparent cure, the history should be investigated for some years. In many cases, when recurrence has taken place, we find chronic appendicitis and practically invalidism as a result.

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[1] Amer. Jour. Med. Sci., Nov., 1908.

If in acute cases there are marked blood changes with high polynuclear count the danger is imminent.

**Treatment.**—The medical treatment can be summed up very briefly. Absolute rest in bed in the dorsal position. The bowel and urine evacuation should occur with the patient confined to bed.

The physician, having made his diagnosis of acute appendicitis, to the exclusion, of course, of general peritonitis, should pursue the following course: All food and even water at first should be prohibited by mouth. Sonnenburg recommends the castor oil treatment of appendicitis, but the writer believes it dangerous. No *cathartic should* at this time be given. Thirst can be relieved by rinsing the mouth with cold water, by small rectal injections of 2 to 3 ounces (60.0–90.0) of hot normal salt solution at 105° to 108° F., or by proctoclysis. If there is much distention, or if nausea or vomiting, lavage should be carefully performed. In these methods I agree with Ochsner. In the following I differ. If the lavage *does not markedly relieve* the distention (if such be present), I would then advise the physician to personally gently wash the bowel with tube and funnel, after the method of lavage. Only about a quart of normal saline in all, at 110° F., need be employed, allowing a few ounces to flow in and then siphoning it out. This practically mechanically carries off the gas and has little influence in producing active peristaltic action. A similar technic is employed in acute distention of typhoid fever with active hemorrhage. He should carefully note the patient's temperature, the rapidity and character of the pulse, and the ratio of pulse to temperature. When feasible, I advocate blood examination in every case, though, of course, it is in some cases impossible. The physician should return to his *case in two to three hours* for the purpose of further examination. The ice-bag should be applied to the painful area at the first visit.

The pain in almost every case can be controlled by the use of an ice-bag of light weight and a small amount of ice therein, so arranged, supported by a circle of gauze, that only the sensitive area is touched by it. A thin layer or two of gauze can be placed between the bag and the skin, so no damage can be done by the cold, which should be continuously applied. The bag can also be suspended from a barrel hoop to take off the weight, but the former method is the simplest.

In emergency I have used bits of ice tied up in dress shields or in pieces of rubber tissue as a substitute for the bag.

On the third visit or the one thereafter (all of which visits should be made within a total period of nine to twelve hours) some definite determination as to *the proper course to follow* at that specific time can be made. Lavage may again be indicated to relieve distention, or even the special intestinal washing, as previously described. Frequently, however, under abstention from food and drink and the application of ice the tympanites will be markedly relieved and physical examination be comparatively easy. If *there is a history of long constipation*, and *on examination a fecal accumulation can be determined* in the cecum, I resort to radical treatment.

Fecal accumulation with gaseous distention is an actual danger by causing pressure on the inflamed appendix. I always resort in such cases to enema or, preferably, gentle intestinal irrigation once or twice a day every day with normal saline solution, with two tubes or a recurrent tube, using 2 to 3 or 4 quarts at each lavage, only ½ pint at a time, at 110° to 115° F., emptying the large intestine mechanically. I have frequently seen the acute symptoms rapidly subside with the above method. These cases are not so very rare.

At the end of forty-eight hours, with symptoms defervescing, a dose of calomel, castor oil, or a saline cathartic is then indicated. If there is vomiting, oxalate of cerium or bismuth and heat to epigastrium; for excessive vomiting, lavage.

The type with fecal impaction is the only class of cases in which such radical methods are resorted to. If in other acute cases in the course of the first ten hours the temperature does not rise markedly, and especially if the pulse does not increase in frequency, but *rather diminishes*, and its character improves, with improvement in the other symptoms, delay, with careful watching of the patient, is allowable. The attack then may gradually entirely subside with no subsequent recurrence, or a fresh attack may take place after an interval. In the case of a first attack, comparatively mild, I would not advise operation subsequent to the attack unless the patient contemplated a journey beyond the reach of a surgeon. Interval operation is always preferable. The indications for surgical intervention are given later.

*Diet.*—As to diet, I am not quite as radical as Ochsner in all cases. For two days in the acute attack, no matter what the type, I allow nothing by mouth, neither food nor water. This checks peristalsis, as Ochsner claims, and furthermore places the patient in the best condition for operation should such suddenly become necessary. The mouth is rinsed and hot salines are given by enema or by proctoclysis. After two days I allow small quantities of hot water by mouth, and if the temperature is 100° F. or over, no food by mouth, but nutritive enemata. As soon as the temperature falls to 100° F. or below, food is given by mouth, in small quantities at first. The general diet should then be fluid for some days; no gaseous fluid should be given. Milk well diluted with lime-water or equal parts with barley-water, oatmeal-water, rice-water, gruels, etc., are excellent. Personally, I believe the latter methods preferable to milk, as causing less tympanites; as the symptoms subside, eggs beaten up with milk, bouillon, chicken broth, and, later, soft-boiled eggs and milk-toast are added.

I agree with Ochsner that lavage is valuable for the distention or for vomiting. If the tympanites, however, is not relieved by the lavage, I believe the funnel method of gentle irrigation of the bowel to be perfectly safe. If fecal accumulation occur later during the attack, a soapsuds enema is indicated. A cathartic should not be given by mouth during the early acute stage until the temperature falls to below 100° F., or particularly until the local symptoms defervesce. Fecal impaction is the exception. Catarrhal colitis should receive treatment as soon as the acute stage of appendicitis has subsided.

Opium has long been a much-vaunted remedy, on the theory of quieting peristalsis and allowing adhesions to form; also for relieving pain; and given preferably as laudanum, or by suppository. I am absolutely opposed to its use. The character of the pulse and respiration are changed thereby; muscular rigidity will relax, and I have seen the symptoms of perforation, both of the appendix and gall-bladder, entirely masked by its employment. Tympanites, distention, and intestinal paresis are more apt to occur as a result of its use. The application of heat[1] I am opposed to in acute cases.

The ice-bag continuously applied possesses all advantages and no disadvantages; and I only advise the use of a single small dose of morphin by hypodermic, and find it but seldom necessary, if the ice does not control the pain.

Frequent examinations of the blood as regards differential leukocytosis and hyperinosis should be made in every case when possible, at first at least twice daily, and thereafter once a day.

If the differential count *is not marked and does not increase*, but *rather diminishes*, and the *symptoms gradually defervesce, do not operate during the acute attack.*

*The indications for operation are as follows:* 1. If the patient shows the symptoms of acute peritonitis when first seen or suddenly develops them—general muscular rigidity, tender abdomen, tympanites, etc.—operate immediately.

2. If there be found on examination an area of resistance in the right iliac fossa, and this increases with more marked symptoms after six to twelve hours' observation, whether chills be present or not, operation is indicated.

An *aspirating needle should never be employed* for purposes of diagnosis.

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[1] It is difficult to secure a continuous high degree of heat and a warm poultice is deleterious.

The blood-count is of value as an aid to prognosis and diagnosis, if the physician has the technical skill or can have it done.

3. In a large abscess, in complicated cases, or when temperature is steadily rising, operate.

4. If the course of the disease is protracted and the symptoms point to abscess or an active and progressive process, operate.

5. In acute fulminating cases. In this type, with apparently mild local symptoms, but especially a *gradual increase of pulse* and a *moderate increase of temperature*, there *should be immediate operation*. The blood examination is important, if possible.

6. A *frequent pulse increasing in rapidity*, not corresponding to the more gradual rise of temperature, *indicates operation*. In all cases when the differential blood-count is marked and increasing, operate.

7. If the patient have a mild attack and subsequently develop a second attack (of less severe type than the first), delay may occur; but if a third attack occur, then an interval operation. If the second attack be more severe than the first, then operate at once.

8. In chronic appendicitis, with symptoms persistent and invalidism occurring as a result, or if recurrent acute exacerbations, operate.

9. In harmful involution of the appendix, operate.

In effect, the best judgment is, operate within twelve to sixteen hours from the onset if improvement does not occur.

The surgeon should operate during the intermediate stage with a spreading inflammatory process, if he unfortunately sees the patient for the first time during this period. A rapid operation with little manipulation is best at this time. In this I disagree with Ochsner, who does not operate at this stage. I continue the other part of his treatment, however.

It is furthermore interesting to note that in C. McWilliams' analysis of 1411 operations upon the appendix at the Presbyterian Hospital, New York, 1 case died following operation for acute appendicitis during the first sixteen hours, and that of pericarditis. Mortality during first twenty-four hours was 5 per cent. Operations on third to sixth day, mortality was 12.7 per cent.; from the seventh to the tenth day, mortality was 20.2 per cent., and thereafter, 13.4 per cent.

The method of operation depends on the location and type of appendicitis.

## CHAPTER XXXI

# DIVERTICULITIS—PERIDIVERTICULITIS

(*Synonyms.*—Sigmoiditis; Perisigmoiditis.)

**History.**—During the past fifty years specimens of false diverticula of the descending colon and sigmoid, both with and without concretions, have been reported by pathologists, who have demonstrated their relationship to general or local peritonitis. Only recently has attention been especially focused on inflammation in the left iliac fossa, and the terms "sigmoiditis" and "perisigmoiditis" been employed.

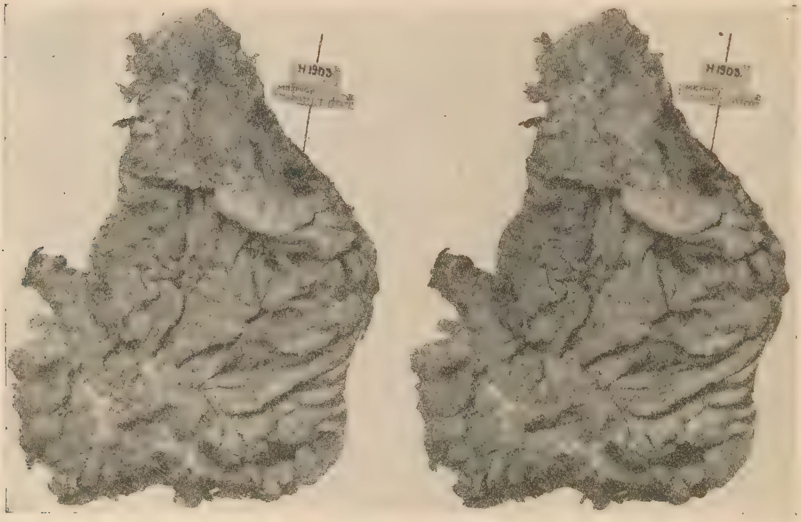

Fig. 282.—Diverticulitis. Sigmoid laid open longitudinally. A diverticulum containing a sloughing ulcer is seen at the lower right hand; another is sectioned near the label needle (W. J. Mayo).

There is confusion as to the definition of sigmoiditis, many using it in the sense of a catarrh of the sigmoid; while others define it as an inflammatory condition, involving to a greater or less degree this entire portion of the gut (the *musculature included*). In other words, it is not a catarrh of the mucous membrane. It was so defined as a diffuse inflammation by Patel[1] and will be so employed in this volume. Specific inflammations, such as dysentery, tuberculosis, and syphilis, are excluded. Eisendrath has recently contributed to this subject.

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[1] Revue de Chirurgie, Oct. and Dec., 1907; Lyon Med., Oct. 2, 1905.

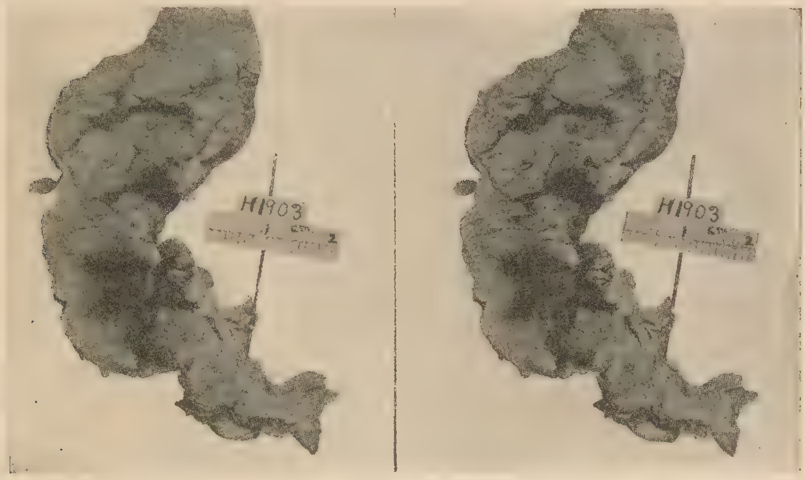

Fig. 283.—Diverticulitis. Section through ulcerated diverticulum shown in Fig. 282 (W. J. Mayo).

Diverticulitis[1] and peridiverticulitis, as when productive of disturbance they occur chiefly in the sigmoid, have been used interchangeably with sigmoiditis; and perisigmoiditis bears the same relationship as does perityphlitis to typhlitis.

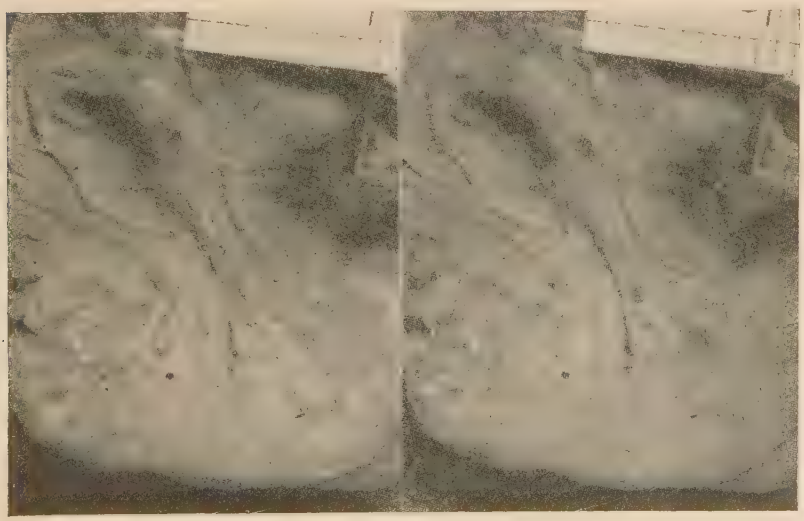

Fig. 284.—Diverticulitis. Enlarged view of sectioned diverticulum shown in Fig. 282. Note muscularis in wall, occluded lumen, and inflamed submucosa (W. J. Mayo).

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[1] Sigmoid Diverticulitis, Archiv. Diag., Oct., 1909.

Mayo first employed the term sigmoiditis, but the condition was first described by Joseph M. Mathews. J. P. Tuttle[1] has recently contributed an excellent monograph. Though inflamed diverticula of the sigmoid undoubtedly cause the maximum of all cases of sigmoiditis and perisigmoiditis, having about the same relation to left iliac abdominal suppuration as the appendix has to similar condi-

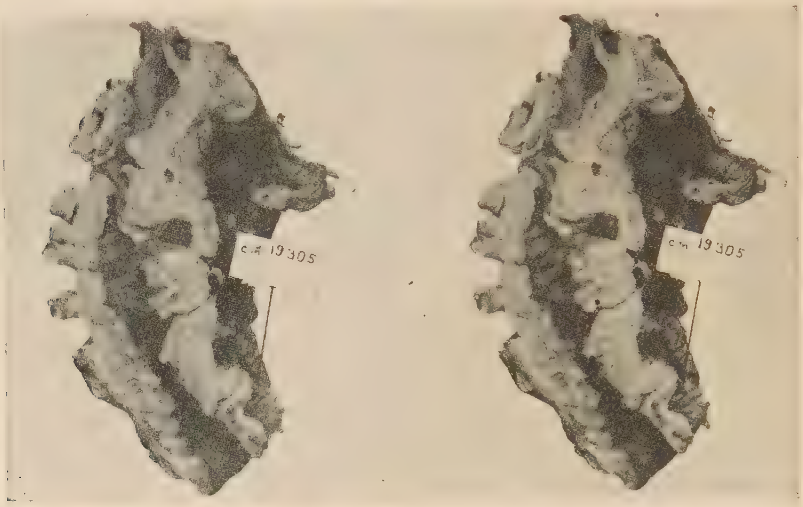

Fig. 285.—Peridiverticulitis. Sigmoid divided longitudinally. Note defective musculature and the diverticula. Inflammatory mass dissected away near label needle (W. J. Mayo).

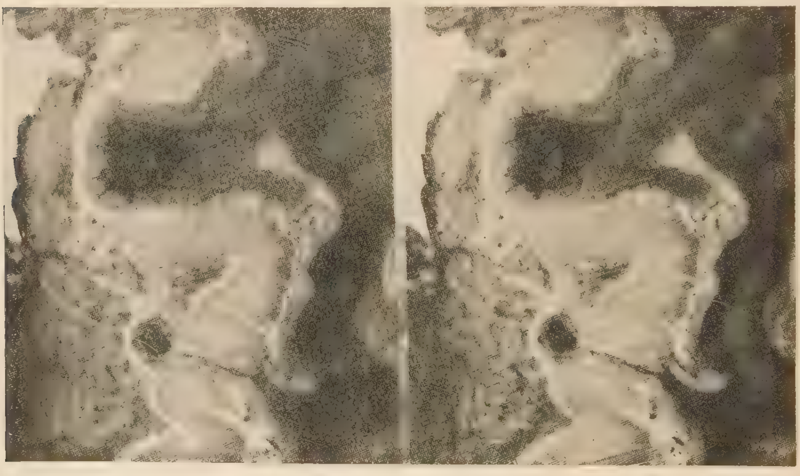

Fig. 286.—Peridiverticulitis. Enlarged view of two diverticula and one point of defective musculature seen in Fig. 285 (W. J. Mayo).

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[1] Amer. Jour. Surg., April, 1909.

tions in the right iliac fossa, yet other causes of perisigmoiditis are given, such as ulcerations extending through the wall of the gut; traumatism or puncture by foreign bodies; diverticula, in association with which Byron Robinson and Tuttle[1] believe that *traumatism* from the iliac and psoas muscles play a part; lumbricoid worms and wisps of hay entering the appendices epiploicæ. Secondary perisigmoiditis is also believed to occur from inflammation by extension from other abdominal or pelvic organs.

Wm. J. Mayo[2] draws a sharp distinction between diverticulitis and peridiverticulitis (Figs. 282–287).

With diverticulitis there is a primary lesion in the mucosa, often of an ulcerative type, and a tendency to perforation into the peritoneal cavity, with resulting acute peritonitis. It has no tendency to produce reduction of the lumen of the bowel.

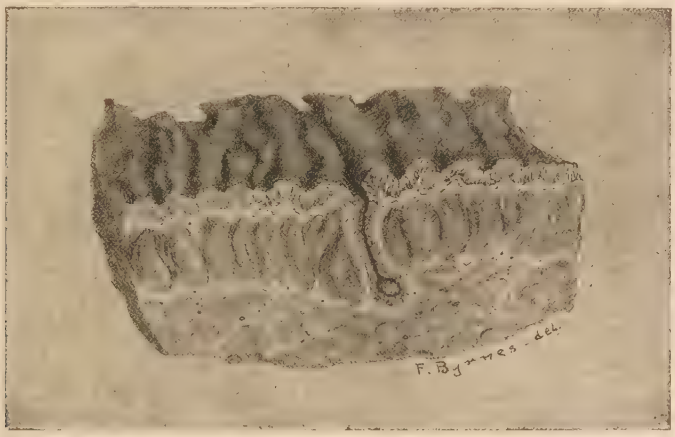

Fig. 287.—Peridiverticulitis. Sketch of diverticulum with inflammatory deposit in subserosa (W. J. Mayo, Wilson, and Giffin).

With peridiverticulitis there is a leakage of toxins and bacteria into the subserosa and tissues surrounding the diverticulum, with resulting inflammation and thickening of the gut, so that its lumen may be markedly reduced and symptoms of obstruction occur. Perforative peritonitis rarely occurs in this type, as so much reparative inflammation goes on. Local intraperitoneal abscess or acute or chronic obstruction may result.

Lejars,[3] Bittorf,[4] and Rosenheim[5] classify sigmoiditis and perisigmoiditis clinically. Gordinier and Sampson[6] hold that these conditions are more frequent than we suppose.

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[1] Amer. Jour. Surg., April, 1909.
[2] Surgery, Gynecology, and Obstetrics, July, 1907.
[3] Semaine Medicale, June 27, 1904, p. 26.
[4] Münch. med. Wochenschr., 1904, p. 147.
[5] Zeits. für Klin. Med., 1904, Band. liv, p. 475.
[6] Jour. Amer. Med. Assoc., 1906, vol. i, p. 1686.

**Diverticula of the Intestines.**—Diverticula are formed by a bulging or protrusion of the intestinal wall, and are divided into the congenital and the acquired.

In the congenital forms the wall of the diverticulum is formed by the whole intestinal wall; and these have been called "true" diverticula. It was formerly thought that all acquired diverticula were of the "false" type, consisting of protrusions of the mucosa through spaces in the muscular coat, so that their wall comprised mucosa and serosa. It has been demonstrated that acquired diverticula may be of the "true" type, and are caused most frequently by traction from tumors or adherent organs. False diverticula are the result of excessive pressure within the intestines combined with a congenital weakness of the bowels. The chief congenital diverticulum of importance is Meckel's.

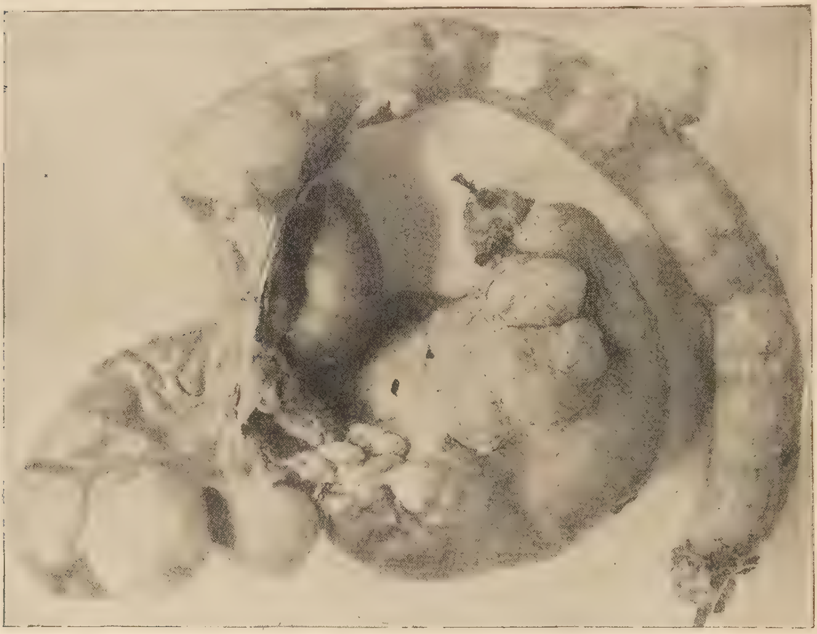
Fig. 288.—Hodenpyl's specimen of multiple acquired diverticula of the colon (sigmoid flexure) (Brewer).

*Meckel's diverticulum*, due to the persistence or incomplete obliteration of the omphalomesenteric duct, usually rises from the ileum $\frac{1}{2}$ to 1 meter above the ileocecal valve, from the convex margin of the intestines opposite the mesenteric attachment, and varies in length from 3 to 10 cm., though rarely longer.

Congenital diverticula have been found in the small and large intestines. An accessory pancreas may be responsible for a diverticulum in the stomach or small intestine.

Acquired diverticula occur both in the small and large intestines, and have even been recorded in the appendix by Edel[1] and Mertius.[2] They are generally more frequent in the large intestine, especially in the lower part of the descending colon and sigmoid flexure, the latter providing the most examples, as in Fig. 288, reported by G. Brewer.[3] Here they are usually multiple.

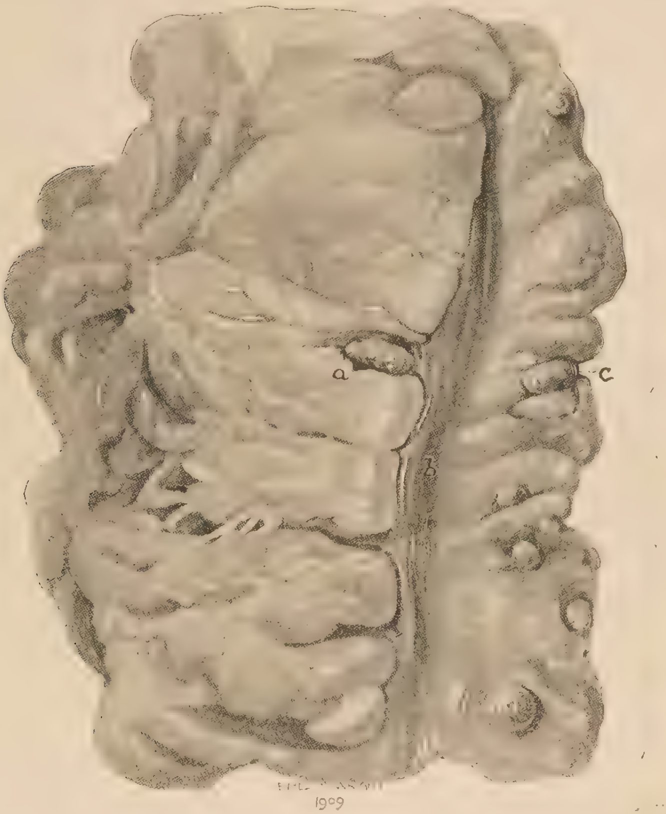

Fig. 289.—Diverticula of sigmoid showing enteroliths *in situ*. The abundant fat has been dissected from off one-half of the bowel, showing the pouches which entered the appendices epiploicæ. At *a* a single pouch has been dissected out and shows well how they are buried in fat and liable to pass unrecognized unless specially sought for; *b*, longitudinal muscular band; *c*, a concretion. (W. H. Maxwell Telling.)

Autopsies in death from other causes have been reported where diverticula were found, but no symptoms had ever occurred. As many as 400 diverticula in one case have been noted by Hausemann.

One case of diverticulitis of the small intestine which gave symptoms has been recorded by Gordinier and Sampson.[4]

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[1] Virchow's Archiv., Bd., cxxxviii.
[2] Mitheilung ans der Grenzgebiet für Med. und Chir., Bd. ix.
[3] Amer. Jour. Med. Sci., Oct., 1907.
[4] Jour. Amer. Med. Assoc., 1906, vol. i, p. 1684.

With diverticula of the appendix, appendicitis has been simulated, but it is clinically unimportant to separate the conditions, as operation is indicated in any event. Diverticula are most frequent in the sigmoid flexure. Quite a number give important symptoms. E. Beer[1] has described the clinical symptoms of diverticulitis, and Telling[2] has collected 105 cases, giving a thorough exposition of the subject.

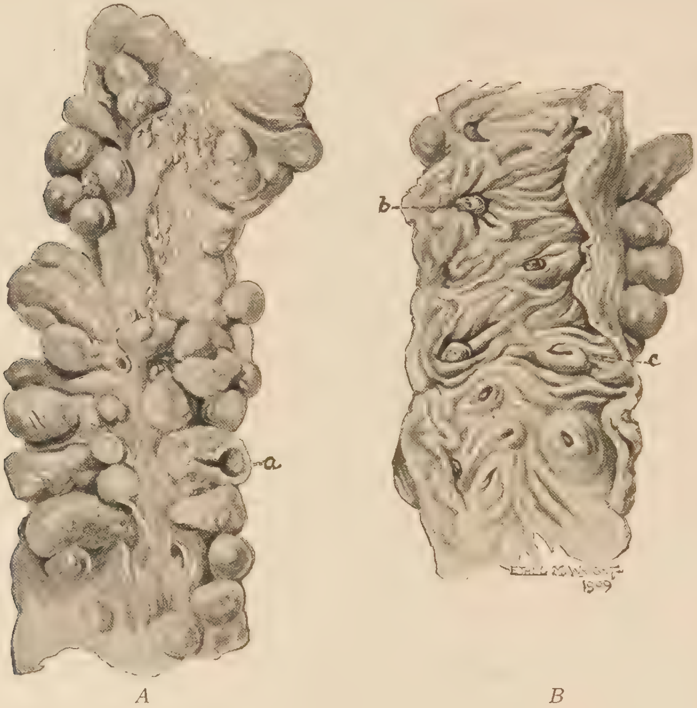

Fig. 290.—Diverticula of sigmoid. *A*, The fat has been dissected from off the outer aspect of the bowel. The pouches are for the most part into the appendices epiploicæ. One of the sacs is laid open at *a*. *B*, Inner surface of the bowel from the same specimen. A concretion is seen at *b* presenting at the orifice of one of the diverticula. At *c*, the lipped orifice is well seen. (W. H. Maxwell Telling.)

Sudsuki[3] found diverticula present 15 times in 40 autopsies, so that the condition is probably more frequent than has been supposed, and has often been overlooked, even in postmortem.

Brewer[4] has reported a case of acute diverticulitis of the sigmoid, with operation before rupture.

**Occurrence.**—Acquired diverticula are much more common in the large intestine, more so in the descending colon, and especially so in the sigmoid flexure. They are usually multiple and may arise from any part of the surface. They are frequently seen in two rows at the sides of the gut, or *close to the mesenteric attachment,* more rarely on the convexity.

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[1] Amer. Jour. Med. Sci., July, 1904.
[2] Lancet, March 21 and 28, 1908.
[3] Langenbeck's Archiv., Bd. lxi, p. 708.
[4] Jour. Amer. Med. Assoc., Aug. 15, 1908.

Chlumsky claims that he finds by experiment that rupture in the living bowel upon distention occurs more frequently opposite the mesentery. The general opinion is that the mesenteric side is less resistant. Probably the most common occurrence of these diverticula is in the appendices epiploicæ. In many cases they are confined to them, and present a double row of symmetrically placed hollowed-out pockets. In other cases none are so situated, and frequently some enter the appendices and some lie outside of them. The presence of the diverticula in these appendices may account for some of the tenderness found in many portions of the gut. The special favoring of the epiploicæ is accounted for by the fact that *the point of their attachment to the gut is a place of least resistance.*

In a majority of cases the affected appendages have been or are filled with a large amount of fat.

Bland-Sutton[1] has stated and illustrated by diagrams that this fat is directly continuous with the subserous fat. If there is the slightest tendency to the formation of diverticula, it will readily be seen that the soft fatty tissue of the appendices epiploicæ form a point of lowered resistance.

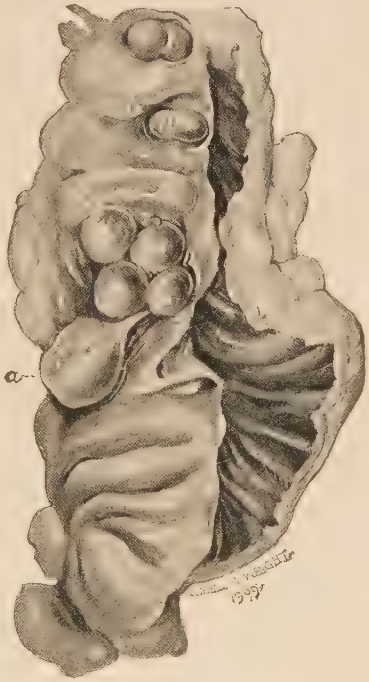

Fig. 291.—Diverticula of sigmoid. The fat has been partly dissected from off the outer surface of the gut, showing several pouches. At *a* is seen a larger one containing a calcareous concretion, with a thin fibrous pedicle in the process of separation to form a loose peritoneal body. A similar concretion, the size of a bean, was free in the pelvis (W. H. Maxwell Telling).

**Size.**—The diverticula vary in size from a millet-seed to a hazelnut. Large size is seldom attained, as secondary changes occur leading to detachment, ulceration, abscess, or peritonitis.

When small, they are semiglobular; as they increase in size, more oval or flask shaped; the aperture on the gut wall usually being smaller than the maximum diameter of the diverticulum, and almost constantly so when they enter the appendices epiploicæ.

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[1] Lancet, Oct. 24, 1903, p. 1148.

This is an anatomic point of great importance in regard to the causation of inflammation. In those which do not enter the appendices the aperture may be relatively large. They are then generally true diverticula and are formed from normal haustra (Sudsuki),[1] usually not extending much above the middle of the descending colon. They increase in number and size from above downward, and may be quite crowded together in the sigmoid flexure. At the commencement of the rectum they generally stop abruptly. This is possibly due to the absence of appendices epiploicæ in this situation; but the fact that the feces are not retained so long in this part of the bowel is also partly responsible. Schreiber[2] thinks that the stronger musculature of the rectum plays some part in preventing their formation.

They are almost invariably filled with fecal material, generally concretions of some degree of firmness. This fact probably determines their subsequent clinical importance, and sharply distinguishes them from *acquired diverticula in the small intestine, which rarely contain fecal material.* To this we can ascribe the immunity of the latter from secondary pathologic processes and symptoms.

Usually in the early stages all the coats of the bowel are represented, but in some the muscularis is absent. There has been controversy as to the presence or absence of muscular fibers in the diverticulum wall. Cruveilhier and Rokitansky originally regarded the diverticula as hernial protrusions of the mucous and serous coats, with absence of muscular tissue. The presence or absence of muscle was for many years the criterion for distinction between congenital and acquired diverticula.

The microscopic findings in numerous cases show that acquired diverticula often *have all the coats of the bowels represented.* In the early stages of the formation it is almost the rule, although the muscular tissue undergoes atrophy as the sac enlarges.

**Etiology.**—One case of an acute catarrhal inflammation of a diverticulum of the sigmoid has been recorded in a child[3] seven years of age, while the great majority are in old or even aged subjects. More recently, J. A. Hartwell[4] and R. L. Cecil have reported a careful study of 18 cases of intestinal diverticula. They find that children and young adults are also subject to them and to the secondary changes occurring in them. They seem to be generally acquired.

1. Generally, advanced age of the patient. In 80 cases the average age was sixty years, but of those in whom diverticula caused symptoms in 47 cases, the age was fifty-five years. In 33 cases no symptoms were referred to their presence, but they were accidentally discovered,

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[1] Langenbeck's Archiv., Band lxi, p. 708.

[2] Deutsch. Archiv. für Klin. Med., 1902, p. 122.

[3] Ann. of Surg., vol. xlvii, by A. P. C. Ashhurst.

[4] Amer. Jour. Med. Sci., Aug., 1910.

the age was sixty-seven. Fiedler[1] records the youngest case at twenty-two years. William J. Mayo places the majority at over fifty years.

2. *Sex.*—In 81 cases—53 males, 28 females.

3. *Obesity.*—Klebs, Mayo, and others have laid stress on this. Many have been fat; the presence of much fatty tissue in the gut walls and appendices epiploicæ have been shown by Bland-Sutton to predispose to mucosal out-pushings. In 22 cases, 17 were stated to be more or less obese and to have much fatty tissue in the gut wall.

4. *Cachexia and Absence of Fat.*—On the other hand, many subjects are noted as having been thin (Hausemann)[2]; 5 of the 22 cases were stated to be of this type. Undoubtedly some had previously been obese, and from age or illness had lost much of their fatty tissue. This has not been definitely referred to. It will be readily understood that the fatty deposit would first weaken the gut wall; the subsequent loss of fat with possible concomitant weakening and wasting of the muscle-fibers would probably increase this tendency.

5. *The Normal Structure of the Large Intestine.*—This readily lends itself to local yielding of its walls, as is suggested by the *normal sacculi, which are often exaggerated in cases of constipation,* sometimes in association with diverticula.

6. *Physiologic Rôle of the Sigmoid Flexure.*—In this portion of the bowel is *the longest retention of fecal material,* and here consequently *the pressure from within will be liable to be the greatest.*

7. *Pressure from Within the Bowel.*—This may be due to accumulation of feces or gas, or both.

(*a*) *Presence of Constipation.*—This stands in *most frequent and important causal relationship to this condition.* In 22 cases in which the point is definitely mentioned, constipation, often severe and generally for a considerable period, was present in 17. In the remaining 5 its absence was particularly noted. Constipation in old people, whether obese or emaciated, *is very common,* while diverticula are comparatively rare; hence, other factors must be present.

(*b*) *Flatulence.*—This is usually associated with constipation, and acts in the same way by *increasing internal pressure;* so much stress has been laid on the factor of internal pressure that the term "*pulsion diverticula*" has been applied to them.

8. *The Relation of the Diverticula to the Points of Entry of the Vessels through the Gut Walls.*—This fact was first pointed out by Klebs[3] in the case of *acquired diverticula in the small intestine,* and has been confirmed by others (Hausemann and Fisher) with regard to the large intestine.

Microscopically it is evident that *the spots in the gut wall* where it is pierced by the *vessels are areas of weakened resistance to internal pressure*, because the vessels are accompanied by a certain amount of lax connective tissue, through which an out-pushing of the mucous membrane can more easily take place.

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[1] Denkschrift der Gesellschaft für Natur. v. Heilknade, Dresden, 1868.

[2] Virchow's Archiv., Band cxliv.

[3] Pathologic Anatomy, 1869, p. 271.

9. *Variations in the Size of the Vessels.*—Graser[1] was the first to investigate this point. In his case, the patient suffered from chronic heart disease, with venous back-pressure, leading to distention of the veins in the gut wall. This dilatation he regarded as further weakening the vessel spaces by pushing aside the muscular fibers. He examined microscopically the sigmoids of 28 patients who had suffered prior to death from mesenteric venous stagnation. In 10 of them he found definite evidence (mostly microscopic) of *commencing out-pushings of the gut wall;* and in every case they occurred through these "vessel holes."

The diverticula were most numerous in the sigmoid and practically ceased at the middle of the ascending colon.

Mesenteric venous congestion may be due to chronic heart or lung disease, portal back-pressure, or intra-abdominal tumor, etc.

"He is inclined to ascribe a special importance to a distention which is not constant, but of frequent repetition, as recurs in the repeated failures of cardiac compensation and in frequent recoveries therefrom in patients with chronic heart disease."

When the vessels are engorged the vessel holes are enlarged; when they are smaller these areas are thereby weakened, and there is a *greater liability to hernia of the mucosa.*

While in the small intestine the diverticula are practically always on the mesenteric side of the bowel where the vessels pierce the walls, *in the larger bowel* many of the diverticula occur on *the side remote from the mesentery.* Another explanation must be sought for.

Therefore, Schreiber believed the congestion of the vessels was secondary to the presence of feces in the diverticulum, rather than to the original cause of the formation of the latter.

10. *The Connective Tissue Around the Vessels.*—Sudsuki[2] found in 40 cases not suffering from mesenteric venous stagnation which he examined microscopically, diverticula present in 15 bodies; in 6 cases, true diverticula, that is, all the coats were therein. In 20 cases, where mesenteric congestion was present, he found diverticula in only 6; in 12 cases free from congestion, diverticula were found in 9.

The subjects were all adults and nearly all men, middle aged or old. He suggests there is *a congenital predisposition with regard to the amount and laxity of connective tissue surrounding the vessels at these spots;* if there is much fatty deposit, this will act in the same way; and if there be subsequent wasting of such fatty tissues, *further weakening takes place.*

Beer states that this theory fails to explain the non-mesenteric diverticula and those which have muscular layers in their walls. These vessel spaces have some influence, but they have some additional cause, and Beer finds this in the following:

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[1] Centralbl. für Chirurg., 1898, etc.

[2] Langenbeck's Archiv., Band lxi, p. 708.

II. *Muscular Deficiency of the Gut Wall.*—Since diverticula occur in old people, in whom the muscular power of their intestines has been more or less exhausted (as evidenced by constipation), and are in association with obesity (or obesity followed by cachexia), these facts all point to a muscular deficiency.

In this muscular weakness Beer thinks the cause of the false diverticula must be sought.

Hartwell[1] believes that an inherent weakness existing in the muscularis of the intestines is probably an important factor in the etiology of diverticula, and in the small intestine this weakness appears to exist along the mesenteric attachment. He reports a number of cases of diverticulitis, and Bruce[2] describes a patient with perforation of a diverticulum, abscess, and adhesions with a secondary appendicitis.

In Mayo's cases areas of muscular deficiency were noted opposite early diverticula, or even in areas yet free from out-pushing. Probably no one factor is sufficient.

Out of 105 cases reported, 60 per cent. were attended with symptoms (Telling).

**Secondary Pathologic Processes in the Diverticula.**—The diverticula of themselves occasion no symptoms, but, as one would expect, readily undergo inflammatory changes therein. They tend in most cases to form fusiform pouches connecting with the lumen of the *gut by a constricting neck,* and these pouches are situated for the most part on that portion of the bowel—the sigmoid flexure—of which the normal anatomy and physiology favor most the retention of feces and the accumulation of gas. If a condition of constipation exists and the " force from within " is increased, it is almost inevitable that they will from the first have fecal contents.

On account of the narrow neck and deficient muscular fibers in the sac wall the contents rarely are expelled and concretions form. These probably cause the trouble. The first result would be a tendency to enlargement of the sac. Then the muscular layers atrophy, as do the glands; the muscle may be replaced by fibrous tissue. The increase in the size of the diverticulum, with atrophic changes in its walls, produces necessarily a dangerous thinning of the sac. In some cases there is little more than a peritoneal covering, with *the contained feces visible through it.* The irritation of the retained and hardened feces then leads to inflammatory changes. These may be slight and only microscopic in the mucosa and submucosa, or may produce *more serious acute or chronic lesions.* The fecal matter is a nidus for bacteria; their products undoubtedly determine the nature of the inflammatory reaction which occurs. An ulceration may result from bacterial infection. Moreover, the concretion will tend to be forced through an inflamed or ulcerated area. These features explain the occurrence of local abscess or general peritonitis. The latter may be also the result of sudden trauma or strain, which may cause an increased pressure within the bowel.

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[1] Ann. Surg., Aug., 1910; Amer. Jour. Med. Sci., Aug., 1910, p. 174.

[2] Ann. Surg., May, 1911.

Definite types are produced by the following *causes* (Telling)[1]:

1. Thinning of the diverticulum wall.
2. Perforating action of the retained concretion.
3. The presence of micro-organisms and their toxins.
4. Inflammatory reaction of varying types and degree.

With these data one can forecast the various cases which one might expect clinically; viz.:

1. Infection of the general peritoneal cavity from thinning of the sac walls without perforation.
2. Acute or gangrenous inflammation—"diverticulitis."
3. Chronic proliferative inflammation, with thickening of the gut wall and stenosis of the bowel—"peridiverticulitis."
4. Formation of adhesions, especially to the (*a*) small intestine, (*b*) bladder.
5. Perforation of diverticula, giving rise to (*a*) general peritonitis, (*b*) local abscess, (*c*) submucous fistulæ of the gut wall, (*d*) fistulous communication with other viscera, especially the bladder.

6. The lodgment of foreign bodies. 7. Chronic mesenteritis of the sigmoid loop. 8. Local chronic peritonitis. 9. Metastatic suppuration. 10. Secondary development of carcinoma. 11. Perforation into a hernial sac. 12. Formation of loose bodies in the peritoneal cavity.

Eisendrath gives a simple clinical classification:

1. Acute: (*a*) Acute catarrhal; (*b*) acute gangrenous, with or without local abscess; (*c*) acute perforative, with general peritonitis.
2. Chronic: (*a*) Chronic hyperplastic or stenosing (peridiverticulitis); (*b*) enterovesical fistulous form; (*c*) chronic adhesive form, causing acute or chronic obstruction.

The following classification, by Telling, of the pathologic processes which result, I believe the most scientific:

1. *Infection of the general peritoneum* as a result of thinning of the sac walls. Organisms make their way through the wall and cause peritonitis *without perforation.* Loomis[2] records 1 case.

2. *Acute or Gangrenous Inflammation of the Diverticulum* (*Acute Diverticulitis*).—*Symptoms.*—Pain, tenderness, and swelling in the left inguinal region are present. Local abscess or general peritonitis may result. Rigidity of the left rectus; fever; hyperinosis (increased fibrin in the blood); leukocytosis; increase in polynuclears are present. If there is general peritonitis, we have the additional symptoms.

[1] The Proctologist, March, 1911.

[2] N. Y. Med. Rec., 1870, vol. iv.

3. *Chronic Inflammation.*—A chronic proliferative inflammation of the submucous and serous coats may occur. Thickening may be considerable. It may lead to (*a*) tumor formation, (*b*) *stenosis with obstruction*, (*c*) *mimicry of carcinoma.*

This type of inflammation is most important, most frequent, and generally overlooked. Grasser[1] in 1898 first described a case. Moynihan recorded one. The mimicry of carcinoma is so perfect that not only is the diagnosis made during life, but also at the operation, and again confirmed erroneously at postmortem (Fig. 292).

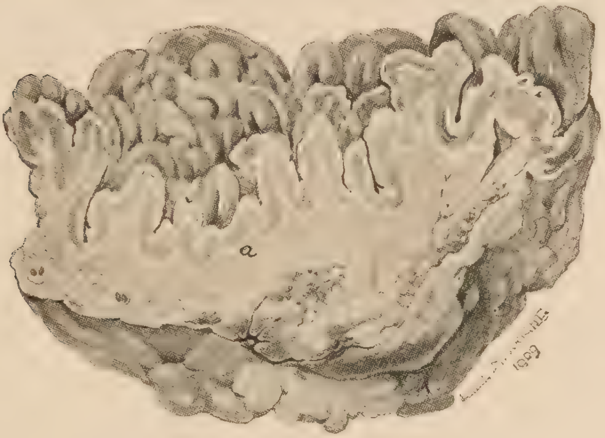

Fig. 292.—Peridiverticulitis with great thickening of the gut wall, causing stenosis and simulating carcinoma, for which it was mistaken when resected at operation. The thickening due to fibrosis is seen at *a* (W. H. Maxwell Telling).

Differential diagnosis between carcinomatous stenosis and diverticular stenosis is as follows:

With *carcinoma* there are nearly invariably an involvement and ulceration of the mucous membrane, with fungation of the growth into the lumen of bowel. With *diverticulitis* it is the rule for the mucous membrane to be free from ulceration (unless a fistulous tract or abscess-cavity open into it from without inward); also the folds of the mucosa are strongly marked and crowded together, giving an unduly rugose appearance (Fig. 293). The orifices of the pouches may be visible, but are generally small and often concealed by these folds. One should examine the folds with a fine probe. There are usually *absence of blood and pus* in the stool in *diverticulitis*, but they are *present* in *carcinoma.*

One must also remember that carcinoma of the sigmoid may originate from diverticulitis.

Age of the patients is usually the same.

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[1] Centralbl. für Chirurg., 1898.

Stenosis may cause acute or chronic obstruction (Mayo).

4. *Formation of Adhesions.*—(*a*) Adhesions to the small intestine may produce acute or chronic obstruction. (*b*) Adhesions to the bladder are also noted.

5. *Perforation of Diverticula.*—Results differ, according to (*a*) acuteness of ulcerative process, (*b*) amount of chronic inflammatory thickening present, (*c*) presence of adhesions.

These factors determine whether perforation leads to (*a*) general peritonitis, (*b*) local abscess formation, (*c*) fistulous communication with other viscera, notably the bladder.

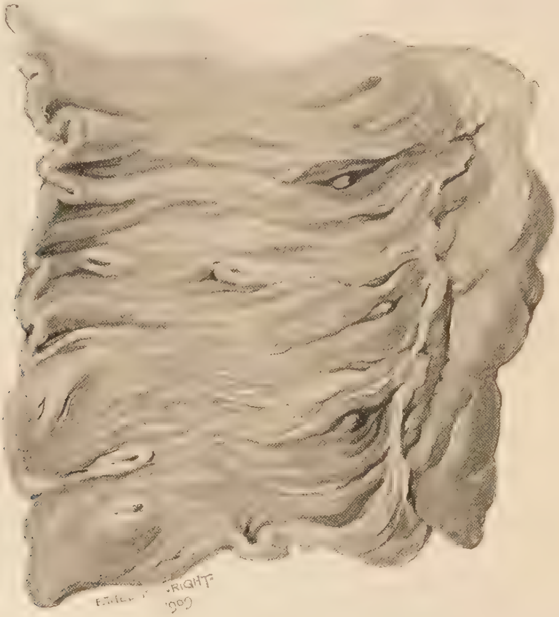

Fig. 293.—Sigmoid showing diverticula. Inner aspect of gut. Normal but rugose mucosa. The orifices show well-marked lipping and concretions protruding partially from some of the pouches (W. H. Maxwell Telling).

(*a*) *General perforative peritonitis* has occurred in 14 cases. C. A. McWilliams reported a case at the Surgical Society, October 30, 1907, upon which he operated at the Presbyterian Hospital for general peritonitis; the history of constipation was of only a week's duration. Male, age forty-seven. The case was believed to be perforative appendicitis, not having been seen *until general peritonitis was in evidence.*

Milky fluid under considerable tension was evacuated at operation. There was also fluid between the liver and diaphragm. Appendix, gall-bladder, stomach, and pancreas were examined, but no perforation was found.

The patient's condition became such that further search was deemed inadvisable. Postmortem showed diverticula of large size commencing in the ascending colon. In the descending colon, 10 cm. below the splenic flexure, a perforation of a diverticulum was discovered. Culture from the peritoneum, etc., showed Bacillus coli.

(*b*) *Local Abscess Formation in 24 Cases.*—There may be several small abscesses shut off by thick adhesions or a single large abscess. The abscess may be intra- or extraperitoneal, and may lead in turn to communication with the external surface, with the bladder or the bowel.

(*c*) *Submucous Fistulæ.*—In some cases with much inflammatory thickening the ulceration of the inside of the sac leads to a small abscess. This happens in several foci in the thickened area. These abscesses tend to burrow through the thickened tissues and form *submucous fistulous* communications with each other. There may be a labyrinth of such tracts. They may re-enter the lumen of the sigmoid or communicate with a peritoneal abscess-cavity by one or several openings. This suppurating process is often excessively chronic and gives rise to *great thickening*, adhesions, and tumor formation, with sequelæ in the shape of intestinal obstruction or adhesions to and subsequent perforation of the bladder.

6. *Lodgment of Foreign Bodies.*—Diverticula are liable to harbor foreign bodies, which may give rise to perforation, diverticulitis, or local abscess. Bland-Sutton reports several cases.

7. *Chronic Mesenteritis.*—In some there is inflammatory thickening of or abscess formation in the sigmoid mesentery. It is believed by Ries[1] that retraction of the sigmoid loop is produced thereby and also adhesions, and hence *twists*, *kinks*, or *volvulus*.

8. *Local Chronic Peritonitis.*—This is often found in the neighborhood of the sigmoid, causing thickening, opacity, or adhesion of the peritoneum, probably in some cases due to leakage of toxins through the thin wall of an overlooked diverticulum, though at times due to the pelvic organs.

9. *Metastatic Suppuration.*—One case recorded, with abscesses of the liver from diverticular abscess (Whyte[2]).

10. *Development of Carcinoma.*—Secondary to diverticulitis.

Carcinoma may result from chronic irritation and ulceration of diverticula due to retained feces (Hochenegg[3]).

11. *Perforation of a Hernial Sac.*—One case is recorded (Stierlin[4]).

**Clinical Aspects.**—Patel,[5] in discussing inflammation of the sigmoid, classifies these conditions as acute non-suppurative sigmoiditis, suppurative perisigmoiditis, chronic perisigmoiditis, sigmoiditis, and sigmoiditis with general peritonitis.

Inflammatory trouble, more or less acute in the left lower abdomen, has been described as left-sided appendicitis, sigmoiditis, perisigmoiditis, epiploicæ appendicitis, diverticulitis, or peridiverticulitis.

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[1] Ann. Surg., vol. xi, p. 523.

[2] Scottish Med. and Surg. Jour., 1906.

[3] Verhandlungen der Deutsche Gesellschaft für Chirurg., Thirty-first Congress, 1902, p. 402.

[4] Correspondenzbl. für Schweitzer Aertze, 1902, vol. xxxii, p. 749.

[5] Revue de Chir., Oct. 10, 1907, and Dec. 10, 1907.

Clinically, the acute cases resemble appendicitis, except for the fact that the inflammation is on the left side. The symptoms are as follows:

Pain; constipation; tenderness on pressure; muscular rigidity, especially of the left rectus, and later tumor; local tympanites is present. Tumor is not always present.

*Left-sided Tumor and Abscess Formation.*—In 63 cases tumor was felt in 20, and abscess formation occurred in 23. In some cases with considerable pus formation there may be but little pain and no pyrexia (Georgi).

Tumor is *elongated, sausage shaped, tender, and often ill defined.* It may be movable or fixed, lying a little above Pouparts's ligament (frequently parallel with it).

Shortly after the occurrence of symptoms, this swelling may be made out; it may disappear rapidly or gradually, or may increase with signs of pus formation. Patel has shown that the tumor may be higher, lower, or posterior. The position depends on the location of the inflamed diverticulum. In a patient of middle age or older the diagnosis of diverticulitis would be probable.

Telling[1] now classifies the diverticular inflammations in the following clinical groups:

1. Inflammatory trouble, more or less acute in the left lower region of the abdomen (so-called left-sided "appendicitis," sigmoiditis, perisigmoiditis, epiploic appendicitis; diverticulitis; peridiverticulitis; pericolitis sinistra, etc.).
2. Perforative peritonitis.
3. Intestinal obstruction: *Acute*, usually due to involvement of the small intestine by an adhesion producing kinking or strangulation by a band. *Chronic*, a mimicry of carcinoma.
4. Vesicocolic fistula.
5. Inflammatory trouble in the right iliac fossa or its neighborhood.

This last has been recently added and will shortly be referred to.

**Differential Diagnosis.**—Differential diagnosis must be made from appendicitis, with left-sided symptoms, pelvic inflammation, ovarian cyst with inflammation or strangulation, actinomycosis of the sigmoid, syphilitic or tuberculous pericolitis, sigmoid catarrh, and dysentery. Tuberculosis is too often accepted. An appendix passing transversely to the left iliac fossa can sometimes be determined by vaginal or rectal examination. Palpation at McBurney's point, examination at Morris' point, and, in addition, of the left lumbar ganglia, may give some information. The determination of the appendix Head zone may also be of assistance. An abnormal position of the cecum occasionally occurs. With tuberculosis, bacilli may be found in the stools, also pus and blood, but not always at first. The tuberculin reaction, ocular or by injection, aids diagnosis. Pus and blood appear in dysentery and syphilis, and the Ameba or Bacillus dysenteriæ in the former and Wassermann's reaction in the latter. Mucus alone is present in catarrh. Actinomyces are found in the stool in actinomycosis. Vaginal examination helps diagnose pelvic conditions.

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[1] The Proctologist, March, 1911.

*Sigmoiditis.*—The writer classifies catarrh of the sigmoid as a colitis (acute or chronic) of catarrhal type, involving this portion of the colon or as "catarrhal sigmoiditis." *True sigmoiditis* Patel defines as a *diffuse inflammation.* In some cases there may be acute inflammatory changes not involving the entire wall of the bowel, which may yield to the external application of ice and enemata. In other cases the entire thickness of the bowel is involved, and there may be local signs with tumor formation, fever, etc. These last cases may be acute, subacute, or chronic. At times the inflammation may subside in any of these types. The writer believes a diverticulum or diverticula are responsible for these cases, and that they correspond to diverticulitis and peridiverticulitis.

*Intestinal Obstruction.*—When acute, this is probably due to secondary involvement of a coil of small intestine, and the diverticulitis frequently cannot be *diagnosed except at operation.*

The same is true of chronic obstruction of the small intestine. Obstruction, which is chronic, recurrent, or acute engrafted on chronic, when localized in the sigmoid region is nearly always diagnosed as the result of carcinoma, when it occurs in an elderly person with a history of constipation.

Advanced age, more recent constipation, cachexia, and blood in the stools favor cancer; while long-standing constipation, absence of blood in the stools after repeated examinations, and slight cachexia (or, rather, loss of weight), together with an evidence of pus formation, would favor peridiverticulitis.

Entire absence of constipation may occur with either, and blood has been found in one diverticular case. Probably many of the so-called cases of cancer are really this condition.

*Perforative Peritonitis.*—In these cases a *routine examination of the sigmoid should be made.* In some cases there may be a previous history of left-sided abdominal inflammation, but in others there is no such history.

*Vesicocolic Fistula.*—Probably many of these cases, supposedly due to cancer of the sigmoid, are the result of diverticulitis with adhesions and perforation of the bladder. Air and feces pass through the urethra. Cripps found 45 cases of vesico-intestinal fistula out of 63 to be inflammatory, only 9 cancerous, and some of these doubtful.

There seems to be a liability of postoperative peritonitis following an operation for diverticulitis, probably from other thin-walled diverticula.

*Inflammatory Lesions on the Right Side of the Abdomen.*—Diverticula may sometimes occur throughout the colon and are found in the cecum. Taylor and Lakin[1] report perforation of a diverticulum 5½ inches from the cecum. This possibility must always be considered; *i. e., in some cases which may simulate an inflamed appendix, the cause may be a diverticulum.*

**Diagnosis.**—To recapitulate: Diverticulitis occurs usually in persons over thirty-five years of age suffering from constipation and of obese habit. The site of pain about corresponds on the left side to that of appendicitis on the right. Muscular rigidity, especially of the left rectus, is present. There is tenderness on pressure in the left iliac fossa. Tumor may or may not be present.

In suppurative cases there may be chills. Leukocytosis is present. The increase in the polynuclears and hyperinosis are of importance for diagnosis.

The various types have been described; among the most common are acute peritonitis, resulting from perforation; local abscess, which may break into some viscus, as the bladder, or which must be opened; acute or chronic obstruction, resulting from stenosis of the sigmoid from peridiverticulitis; chronic tumors, simulating carcinoma; and milder cases, with local pain, tenderness, constipation; and a local tumor, which symptoms gradually subside under treatment.

**Treatment.**—The acute cases should receive the same treatment as *acute appendicitis*. In mild cases the bowel should be emptied by enema; liquid diet, preferably broths and gruels; ice-bag continuously applied; absolute rest in bed. Subsequent *attention, after recovery*, should be directed to the careful regulation of the bowels by diet and medication. Intestinal irrigation and high olive oil injections also are of value. Cases of chronic stenosis may be temporarily treated as such. Indications for operation are the same as in appendicitis.

Acute obstruction, peritonitis, and abscess require immediate operation. Chronic obstruction may require resection. Appropriate operative procedures are necessary for the complications described.

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[1] Lancet, Feb. 19, 1910, p. 495.

## CHAPTER XXXII

# INTESTINAL OBSTRUCTION—ACUTE AND CHRONIC

INTESTINAL obstruction occurs in two types—acute and chronic.

Acute obstruction is, in turn, characterized by two anatomic types:

1. Acute intestinal obstruction with sudden complete occlusion of the intestinal lumen.

2. Acute intestinal obstruction engrafted suddenly on a chronic obstruction (chronic stenosis), due to a sudden blocking of the stenosed intestines from various causes, such as by a foreign body or a fecal accumulation above the stricture.

## ACUTE INTESTINAL OBSTRUCTION

(*Synonyms.*—Ileus; Miserere; Passio Iliaca.)

**General Considerations.**—Acute intestinal obstruction may be defined as a sudden acute stoppage of the passage of the intestinal contents. This may be caused by mechanical occlusion of the intestinal canal (mechanical ileus), by a sudden loss of motor power in a portion or in all the bowel (dynamic or paralytic ileus), or by a combination of these conditions.

Before discussing the matter further, as a means of assistance to diagnosis, I wish to call a few very important facts to my readers' attention.

As a rule, we may say that acute obstruction of the small intestine gives rise to more severe and violent symptoms than that of the large intestine. The nervous apparatus of the small intestine is in connection with most important plexuses, the solar and superior mesenteric, and the pain is more violent, the vomiting earlier and more marked, and the prostration and shock more rapid.

*The early appearance of indicanuria and of anuria are also significant of obstruction in the small intestine.*

Moreover, simple occlusion of the intestines does not lead to nearly as acute symptoms as when strangulation is present. In the latter condition interference with the circulation of the intestinal wall and of the mesentery and the irritation of the sensory nerves give rise to acute and violent symptoms, marked pain, vomiting, and shock. In this class of cases we have the acute internal hernias and strangulations, volvulus, and the severe type of acute intussusception. Local meteorism is generally present in the early stages of volvulus, internal herniaform strangulation and kinking of the bowels, and enables us at times to locate the character of the lesion. Often this distention rapidly becomes very extensive, as in volvulus, for example, a general distention ensues, so that determination of the condition is difficult.

In the majority of cases of acute obstruction when we find present marked increased peristaltic movements of the bowel, with stiffening and rigidity of loops of intestines; they occur in acute cases engrafted on cases of chronic stenosis with hypertrophy of the bowel muscle above the stricture. We occasionally see this symptom in primary acute conditions, especially in acute intussusception.

We must remember, moreover, that acute dilatation of the stomach, either alone or associated with intestinal paresis, quite frequently occurs after operation, and presents many of the symptoms of acute dynamic ileus. There is obstruction, in fact, in many cases due to pressure of the organ on the transverse duodenum or, as some believe, by mesenteric traction. The stomach may occupy the left half, or even the entire abdominal cavity. I have referred to this condition under Acute Dilatation of the Stomach.

Practically we may say that in every case of acute obstruction, intra-abdominal tension is markedly increased, thus interfering with the physical examination. I place the following suggestions at the commencement of this chapter in order to emphasize them the more. They are of value as an aid to diagnosis in every case with acute symptoms.

Immediate and thorough lavage, digital examination of the rectum, and if no evidences therein of obstruction or intussusception, preventing the entrance of an injection, then a careful recurrent rectal irrigation; vaginal examination and inspection of hernial openings should be the preliminaries in the examination of every case. Lavage and irrigation of the bowel immediately promote the comfort of the patient by lessening distention, render the physical examination easier, and the combined methods are thus an invaluable aid to diagnosis. It may be necessary to substitute a high enema for irrigation.

**Etiology of Acute Intestinal Obstruction.**—The various causes of acute intestinal obstruction, on account of their importance, will now be considered separately:

The first classification that I shall describe is the so-called *internal herniaform strangulation of the bowel* (compression of the intestines), due to strangulation by (*a*) bands and adhesions, the result of a former peritonitis; (*b*) Meckel's diverticulum; (*c*) slits and apertures; (*d*) incarceration into herniæ; (*e*) tumor pressure from without.

*Frequency.*—Fitz, in an analysis of 295 cases of acute obstruction, gives 34 per cent. of the cases as due to this type of strangulation (internal herniaform), excluding volvulus; 35 per cent. out of 1134 cases are reported by Leichtenstern.

Out of 101 cases of strangulation Fitz shows that 63 were due to adhesions and bands, and 21 to vitelline remains.

Strangulation of the intestines by adhesions and bands thus constitutes the largest percentage of this class. In some cases they may be congenital. The band may be a firm fibrous cord, or may be tough and thin as a thread, occasionally it may be ½ inch wide.

Strangulation from bands and adhesions may occur in four ways:

1. There may be a short tense band firmly attached at each end, beneath whose arch a knuckle of the intestines passes, the space may only be of a size to admit two to three fingers.

2. On the other hand, there may be a long lax band, attached at its ends and forming a ring or spiral 8, through which a loop of the small intestine may slip.

3. A loop of intestines filled with contents may lie over a tense band of adhesions and thus become strangulated; this is a rare occurrence, but has been described by Treves.

4. The intestines may suddenly become kinked and occluded by traction from an adhesive band, as if an ovarian cyst were tapped and the sudden contraction drew on an adhesion to the intestines.

These bands and adhesions may occur between any of the viscera, the parietal peritoneum, omentum, and mesentery.

Adhesions may surround the bowel and contract, narrowing its lumen, also a mesenteric contraction may be a cause.

A coil may be caught between the pedicle of a tumor and the pelvic wall or may circumscribe a tumor pedicle.

*Strangulation by Meckel's Diverticulum.*—Meckel's diverticulum is due to the incomplete obliteration of the vitelline duct, and forms a finger-like projection from the ileum, usually within 18 inches from the ileocecal valve. As a rule, it is about 3 inches long, though frequently longer, and cylindric in shape with a conic end, though the latter is occasionally "clubbed." The end may become attached to the abdominal wall near the navel, to the mesentery, or to some other point, and thus form a band or loop under which strangulation may occur. More often the diverticulum is free and may form a ring into which its end projects. A loop of the intestines may enter the ring and, especially if the tip is club shaped, may push it before it and tie a knot (Fig. 294).

The vermiform appendix may become adherent to some point in the peritoneal cavity and form an arch under which a loop of the intestines may become strangulated. This may likewise occur with the Fallopian tube.

*Strangulation of the Bowel Through Slits and Apertures.*—This type is less often met with, and is, in fact, quite rare. Slits and apertures in the mesentery and omentum may be congenital, but are more frequently traumatic. They generally occur in the mesentery near the lower part of the ileum. Fissures, holes, or rings are more frequently formed by peritoneal adhesions; more rarely strangulation may occur in a tear of the uterus or bladder.

*Strangulation from Internal Herniæ.*—These are situated within the abdominal or thoracic cavity, or are subperitoneal or retroperitoneal, parallel to the abdominal wall without passing outward.

In some of the so-called external herniæ no swelling can be detected externally. Moreover, hernia may develop in an accessory form between the muscles and fascia.

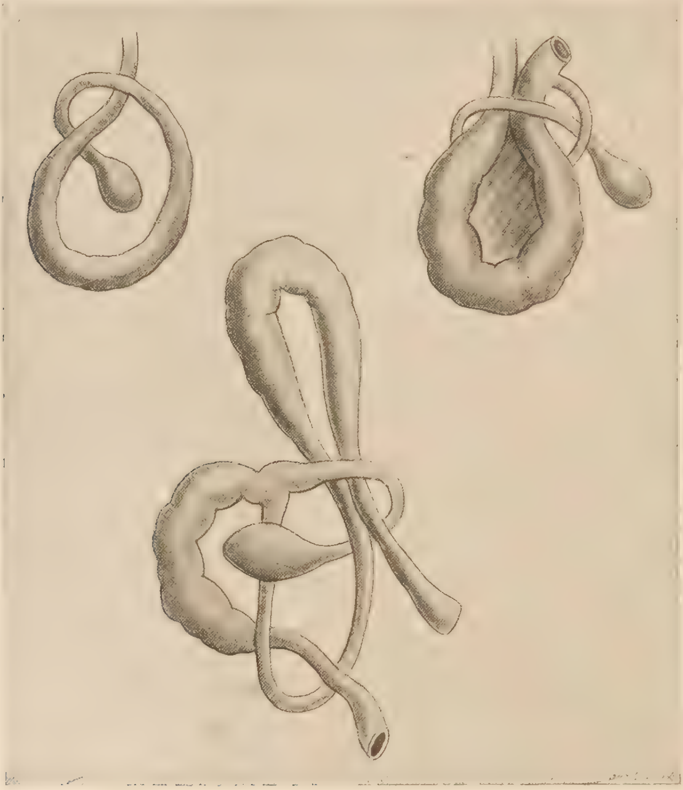

Fig. 294.—Knotting of a Meckel's diverticulum which has a button-like swelling of its extremity (after Treves).

Among internal herniæ we may have those of the recessus duodenojejunalis, intersigmoid recess, retroperitoneal, anterior, retrocecal, foramen of Winslow, and diaphragmatic (Figs. 295–297).

Diaphragmatic hernia is most frequently met with of these forms and can occasionally be diagnosed.

It may be congenital or traumatic, as from wounds, contusions, or excessive vomiting.

In the true form, a hernial sac of peritoneum or pleura covers the viscera; in the false form (the more frequent) it does not. This type is found on the left side, the stomach being involved most frequently, the colon and small intestine less so.

The stomach fundus passes into the thorax and apical rotation occurs.

*Physical Signs of Diaphragmatic Hernia.*—Pneumothorax, less motility of the side of thorax involved, bulging of the walls of the thorax, and metallic sounds are the chief symptoms. Dyspnea, distress or pain in the thorax, and difficulty in swallowing (dysphagia) may be present. Diaphragmatic hernia can be diagnosed by inflation of the stomach with air or water, the use of the *x*-ray, or by gastrodiaphany.

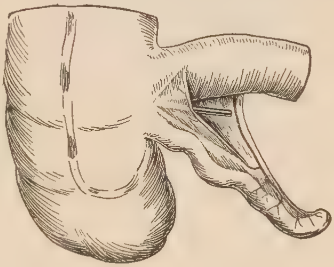

Fig. 295.—A rod in the ileo-appendicular, or ileocecal, fossa (Cantlie).

*Mechanism of Internal Hernia-form Strangulation (Compression of the Intestines).*—The *mechanism* of the type of obstruction just described is as follows:

A coil of gut is drawn beneath a band or through an aperture, and becomes at once strangulated, or congestion of the mesenteric vessel occurs, then gas and pus accumulate, and later strangulation occurs, or, in addition, a torsion of the bowel may take place—necrosis, gangrene, and perforation result.

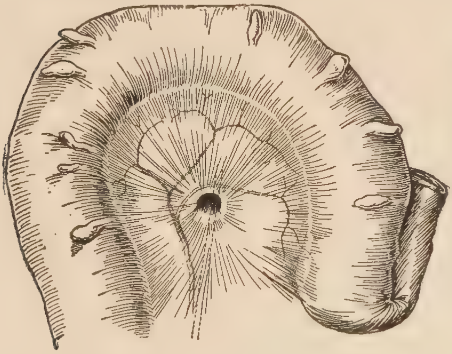

Fig. 296.—The intersigmoid fossa exposed by holding upward the pelvic colon; the double dotted line indicates the ureter behind the peritoneum (Cantlie).

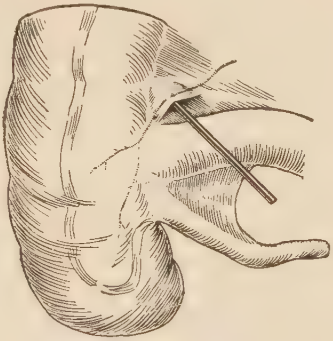

Fig. 297.—A rod in the ileocolic fossa (Cantlie).

The lower part of the ileum is chiefly affected. Peritonitis may occur early, in twenty-four hours, or late, in one to two weeks. Various types of strangulation are shown in Figs. 298–305.

*Sex and Occurrence.*—In males 70 per cent., though some claim occurrence in the sexes is equal. Between twenty and forty years cases chiefly occur, though 40 per cent. are between fifteen and thirty

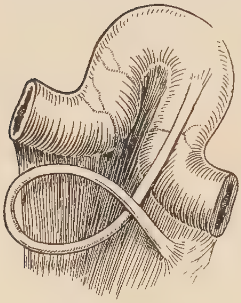

Fig. 298.—A band in the form of a loop attached to the intestine and mesentery (Cantlie).

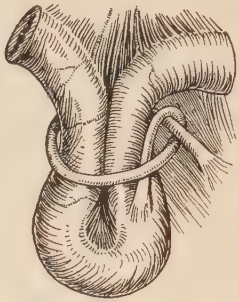

Fig. 299.—The coil of intestine has slipped through the loop of Fig. 298, and caused strangulation by a complicated knot (Cantlie).

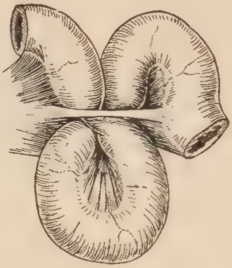

Fig. 300.—A coil of intestine, in addition to being snared under a band, has undergone rotation on its mesenteric axis (Cantlie).

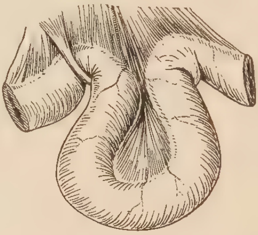

Fig. 301.—A knuckle of intestine snared over a band which is stretching from the intestine to the mesentery (Cantlie).

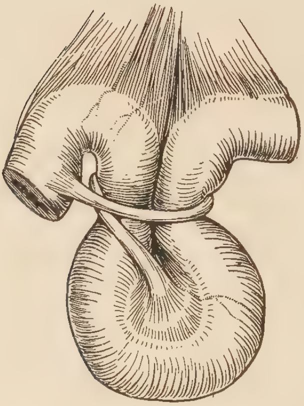

Fig. 302.—A knuckle of intestine snared by a band which has assumed the form of a simple loop (Cantlie).

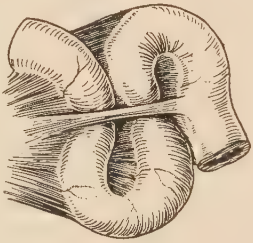

Fig. 303.—A knuckle of intestine snared under a band which is stretching from the intestine to the mesentery (Cantlie).

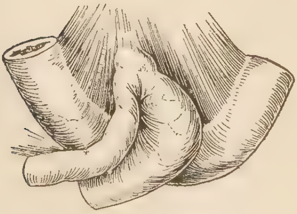

Fig. 304.—A coil of small intestine adherent to a caseating tuberculous mesenteric gland (Cantlie).

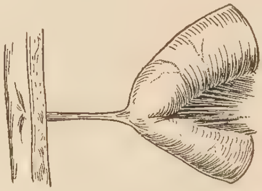

Fig. 305.—A band attached to the small intestine and umbilicus, causing an acute kink of the bowel (Cantlie).

years. In 90 per cent. the seat of the trouble is in the small intestine, usually the ileum; in 67 per cent. strangulation is in the right iliac fossa; in 83 per cent., in the lower abdomen (Osler).

**Clinical Symptoms.**—These are characterized by their sudden acute onset while the patient is in perfect health. Rarely injury, violent movement, or diarrhea may precede the attack, and are considered to be factors. Sudden severe pain first occurs, occasionally colicky, often severe throughout, though at the end it may lessen. The pain at times corresponds to the seat of the strangulation. Vomiting begins early and is persistent and soon becomes feculent; *absolute constipation* is present, no flatus is passed. There is early and rapid appearance of collapse, urine is scanty, meterosm slight or absent, slight tenderness on pressure, no blood in bowel movements if such occur, nor is it discoverable by enema. Death occurs in two to four days.

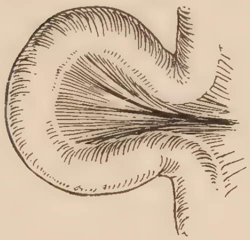

Fig. 306.—Illustrating the disposition of a loop of bowel which is predisposed to volvulus. The parietal attachment of the mesentery is considerably shorter than its intestinal attachment, and the coil is, as it were, suspended as from a pedicle (Cantlie).

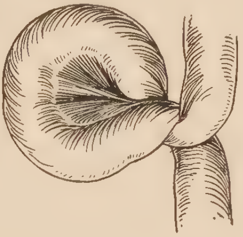

Fig. 307.—Illustrating a volvulus in which the upper segment of intestine passes in front of the lower one (Cantlie).

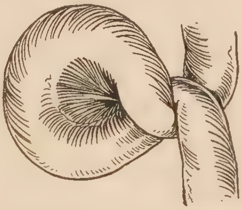

Fig. 308.—Illustrating a volvulus in which the upper segment of intestine passes behind the lower one (Cantlie).

If peritonitis occurs, meteorism is marked and pain recurs and is severe. A circumscribed area of dulness or tumor is rare. The attack is of a fulminating character.

**Volvulus** may be defined as an obstruction of the bowel caused by a rotation of the intestines about their axis, a rotation of the bowel about its mesentery, or an intertwining (rotation) or knotting of two intestinal loops with their mesenteries.

It occurs most frequently in the sigmoid flexure. The ascending colon, cecum, and small intestine, especially the ileum, may sometimes be affected, and intertwining of the intestines is most often met with in this location.

*Mechanics.—The Sigmoid Flexure.*—The sigmoid flexure must be large and its mesocolon long and narrow for the development of volvulus. The ends of the sigmoid are thus approximated, and it can readily rotate around the mesocolonas a pedicle (Fig. 306). This condition may be congenital or due to some chronic inflammation.

*Etiology.*—Chronic habitual constipation is the *chief cause*, as the weight of the fecal matter produces local displacement and distorts and elongates the mesentery.

The rotation is usually the result of bodily exertion. On the other hand, one branch of the flexure, being overdistended, may drop down over the other. The branches may be rotated through 180 to 360 degrees, or even several complete rotations may occur. The distention of the sigmoid, the congestion and exudation of fluid and accumulation of gas in the loop, prevent restitution to the normal position. The accumulation of gas in the colon also prevents return to normal. Valvulus is illustrated in Figs. 307–308.

*Small Intestine.*—The same conditions favor axial rotation of the small intestine.

Inflammation of the mesentery is sometimes a cause, also gallstones, by producing colic and spasm.

*Accessory Causes.*—Leanness, the absence of fat in the mesentery, and hence lessened intra-abdominal pressure have been considered predisposing causes, also trauma, jumping and lifting, sudden diarrhea (peristalsis) in these constipated cases, or a large enema (according to some).

*Age.*—It is more common in older than in young subjects, generally in those over forty years of age, though Fitz places it more frequently at thirty to forty years in his statistics.

*Frequency.*—Fitz finds 42 out of 295 cases of acute intestinal obstruction due to volvulus.

*Sex.*—According to Fitz it is more frequent in men—68 per cent. Some place the proportion much higher, as 3 or 4 men to 1 woman.

*Symptoms.*—This condition is characterized by its acuteness and rapid course. It is more rapid than any other form, except, possibly, internal strangulation of the intestines.

*Pain* is sudden, violent, and never absent, and sometimes localized in the left lower quadrant; it may remit, but there is never a complete intermission. Vomiting is, as a rule, violent and profuse, continuous, and is an early symptom, though occasionally less frequent in sigmoid volvulus. *Fecal vomiting is comparatively rare. Constipation,* both for *feces* and *flatus*, occurs, as a rule, from the incipiency of the attack. Occasionally a history of violent exercise or of acute diarrhea may precede the attack. Tenesmus may be present in volvulus of the sigmoid, though not as frequent as in intussusception, and in rare instances there is the passage of a small amount of blood.

Local meteorism is an important symptom, *the left lower portion of the abdomen* protrudes as a tense and elastic swelling, feeling like a rubber ball; visible peristaltic movements do not occur; percussion may be tympanitic, there may be metallic sounds or, rarely, dulness if there is much edema. In some cases there is an S-shaped protrusion of the abdomen, most prominent in the upper left and lower right quadrant. This is in the early stages. General meteorism develops most rapidly, *next in rapidity to that of general peritonitis*, but in the latter the abdomen is very tender; while in the former it is only slightly so. Within forty-eight hours the abdomen may be balloon shaped. This *rapid meteorism is an aid to diagnosis.*

The symptoms are very acute and collapse is marked. In axial rotation of the small intestine at the onset there may not be absolute constipation; the local meteorism starts higher up and there is no tenesmus. In these cases, curiously enough, the symptoms are no more violent than in sigmoid volvulus.

*Anatomy.*—There is local meteorism, the walls of the distended loop are thickened, rigid, edematous, and dark red. The peritoneal coat is often torn, as is the muscularis. Hemorrhages are seen in the intestinal wall. Blood, gas, mucus, and feces lie in the loop. The mesentery is hyperemic and infiltrated. The twisted part of the intestines is attenuated and pale. Gangrene may occur at the line of demarcation. Other parts of the intestines are collapsed and pale unless general meteorism subsequently occurs.

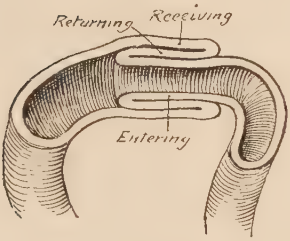

Fig. 309.—Intussusception.

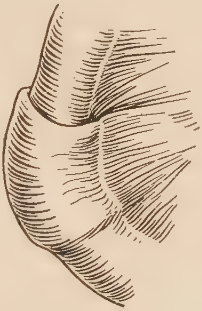

Fig. 310.—An intussusception (Cantlie).

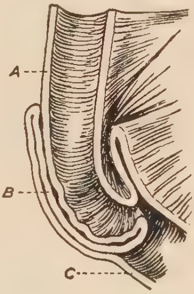

Fig. 311.—An intussusception in longitudinal section: *A*, The entering layer; *B*, the returning layer; *C*, the sheath (Cantlie).

*Course.*—The course of volvulus is always acute. The patients may occasionally die in twelve to twenty-four hours or in two to three days; the average course is a week. They die of collapse, exhaustion, peritonitis, or from paralysis of the heart due to compression from tympanites. Unless operation is performed, the cases, as a rule, are fatal, especially those with complete axial rotation.

Some patients who, from the symptoms, seem to have suffered from partial rotation, the course being *more* chronic, have recovered.

**Intussusception; Invagination.**—It is in this condition that one segment of the intestines slips into an adjacent segment, so that the latter forms a sheath for the former. The two portions make a cylindric tumor which varies in length from an inch to many feet. The condition is always a descending intussusception. The outer tube is called the sheath or intussuscipiens; the middle and inner tubes, the intussusceptum; the innermost tube, the entering tube; the middle one, the returning tube (Figs. 309–311). The upper part, where the middle tube bends over into the sheath, is called the neck; its lower part, where the inner tube bends over into the returning cylinder, is called the head. Mucous membrane is in contact with mucous membrane and peritoneum with peritoneum. Those depicted are the usual form, sometimes a double or triple invagination may occur, or an incomplete lateral invagination, the reverse of a diverticulum. The mesentery participates in the invagination and becomes compressed and wedged in the sheath. The serous surfaces may become adherent, so that the invagination cannot be disengaged. There are various types of intussusception which may occur in every portion of the bowel:

1. *The Ileocecal Form.*—In this type the ileocecal valve forms the apex of the intussusception, the ileum the internal cylinder, and the colon the sheath; this often forms a very long intussusception, so that the valve may even protrude at the anus.

2. *The Ileocolic Form.*—The lowest part of the ileum forms the apex and protrudes into the cecum. If the cylinders become adherent, then the ileocecal valve and cecum may become inverted.

3. *Ileaca-ileocolic Form.*—There is first an ileac invagination which may force the intussuscepted part into the colon.

There may also be intussusception of the ileum, jejunum and ileum, jejunum, duodenum and jejunum, duodenum, colon, colon and rectum, and, rarely, of the rectum.

The ileocecal form seems to predominate, and is especially frequent in children.

The ileocolic is also quite frequent, and these two types give the longest intussusceptions.

In adults the ileac and ileocecal forms occur with about equal frequency, and in the colon it is quite frequent.

Intussusception of the vermiform appendix into the cecum has been reported, and also of the ileum into the open Meckel's diverticulum, when this is patent at the umbilicus, and also by the gut, grasping it, or by traction.

The mesentery exerts traction on the intussusception and the bowel curves so that the concavity points toward the mesenteric attachment. This may cause a kinking, which further closes the intestinal lumen. Circulatory changes take place in the intussusception, edema, swelling, etc., and in severe cases strangulation and gangrene. The intussusception may slough off. Peritonitis is first noted on the second or third day; it may be local or become general.

*Mechanism of Intussusception.*—The probable reason of intussusception being so frequent in young children is that during the early months of the infant's life there is a rapidly increasing disproportion between the transverse diameters of the large and small intestines, the large intestine increasing very rapidly in diameter, so that the ileum can readily prolapse. For the production of invagination there is probably a local spasm of a portion of the intestines, and the normal gut below is pulled upward by its longitudinal fibers over the contracted piece of bowel, and the irritation caused by the invaginated part then causes spasmodic contraction of the gut above, which carries the incarceration further downward.

Numerous experiments have been carried out for a study of the mechanism. None of these seem to show that primary paralysis is a factor, but that the condition is rather of a spasmodic type.

Paralysis of a limited part of the bowel may, I believe, be a factor in some cases.

*Frequency.*—Weiss finds that out of 321 cases, intussusception occurs:

| | Per cent. | Per cent. | Per cent. | Per cent. |
|---|---|---|---|---|
| In the newborn and sucklings | Iliac, 24 | Ileocecal, 42 | Ileocolic, 10 | Colic, 24 |
| Childhood to puberty | Iliac, 23 | Ileocecal, 43 | Ileocolic, 14 | Colic, 26 |
| Adult | Iliac, 29.5 | Ileocecal, 34.5 | Ileocolic, 4.5 | Colic, 27 |
| Meckel's diverticulum (adult). | 4.5 | | | |

177 in first year; 85, two to fourteen years; 59, later age.

The agonal type of intussusception occurs just before death, is of no importance, and is often multiple. Of Leichtenstern's cases, 131 out of 543 occurred in the first year, 80 of them at four to six months.

*Age.*—Most frequent in infancy and early childhood, up to the fifth year.

*Causes.*—Diarrhea, intestinal polypi, carcinoma and stricture, ingestion of irritating food, contusion of the abdomen, shaking the body, and acute and chronic diseases have been given as factors.

Benign tumors when present, such as accessory pancreas, fibroma, myoma, especially the polypoid form, are generally at the apex of the intussusception, and most frequently occur in the ileum. They may set up peristalsis, and constriction and invagination so result.

It is rare with carcinoma or stenosis, and it may occur when the tumor is pedunculated. Often the cause is not discoverable.

*Symptoms.*—In the acute cases the attack is characterized by its suddenness. There may be preliminary intestinal disturbances, such as diarrhea or colicky pains. Often they appear while the patient is quiet, asleep or nursing. I shall refer to the acute cases only in this chapter. The iliac and ileocecal forms are the most acute. The colic and rectal form are more gradual.

*Pain.*—There is first pain of a violent colicky character, at times arising at a definite point. It may be very severe and overwhelm the patient, and in children may cause convulsions, or they may scream and groan. It is continuous at first, later may intermit, though at times it may be continuous throughout. In some cases the pain is in the right iliac region, or occasionally at the umbilicus, and at times local tenderness is present. Spasms and contraction with rigidity of loops of intestines sometimes occur in acute cases, but more usually in the chronic. Vomiting is constant and early in children, is not constant in adults, and hence is a less important symptom in them.

The vomiting depends on the position of the intussusception; the lower down it is, the less likely the vomiting at the outset; peritonitis brings it on.

Feculent movements may occur at first; later, diarrhea with blood and mucus, and tenesmus if the invagination is low down. In some cases this is quite marked; *hemorrhages* from the *bowels ensue*, even if the invagination is high up.

*Fecal vomiting is rare.* Vomitus usually contains gastric contents, mucus, and bile, and only occurs in one-fourth of the cases in adults. The higher up the invagination, the sooner it appears. Meteorism of a great degree is exceptionally present; sometimes the abdomen is even retracted. It is dependent on the degree of constriction.

*Tumor.*—A palpable tumor can at times be appreciated in about one-half the cases. It varies in size, may seem shorter to the touch than it really is, or it may impart the feel of a double swelling; it is usually of a cylindric or sausage shape, elongated, and curved. Its consistency may vary at different times and it can be slightly compressed. It may feel hard and then suddenly soft, or even vanish. The changes in *consistency and resistance* are due to the spasmodic *contraction* in the intestinal wall. During contraction it may be tender.

*Situation.*—Most frequently the tumor is located in the region of the sigmoid; then at the anus or rectum, then at the cecum, descending transverse or ascending colon.

The position of the tumor may be permanent if there are any adhesions, or it may change if the intussusception increases.

Prolapse of the swelling through the anus often occurs.

*Duration.*—Especially in children severe symptoms of collapse may take place within two to three days, and if vomiting persists, it may be feculent.

About 80 per cent. of young subjects die; subacute cases run two to four weeks and spontaneous cures occur in this type.

Death from collapse, gangrene, and peritonitis may occur during the first week in infants, or in two to three weeks in adults. Sepsis and phlebitis may be associated.

*Diagnosis.*—Acute commencement, vomiting constant in children, but not so in adults; at times tenesmus, the *presence of bloody stools;* then retention of feces and flatus, *distention of the abdomen* usually slight; and last the appearance of a tumor are diagnostic of intussusception.

*Intussusception of the Appendix.*—Moschowitz,[1] reports a case of intussusception of the appendix (Fig. 312), and collects 24 additional cases. In the pure uncomplicated cases, without invagination of the ileum into the cecum, the history usually covers a considerable period from a few weeks to even several months. The oldest patient was forty-two years old, the youngest two, and the general average was five years of age.

*Symptoms.*—The pain is usually intense, cramp-like, and localized around the umbilicus. It occurs in paroxysms, during which period the patient suffers severely. The distinguishing feature of the pain is its remitting character. During the period of remission the patient may apparently feel perfectly well. Attacks of pain with remission alternate and there may be from one to two to many attacks a day. The duration of the disease varies from a few weeks to several months. There is rigidity of the abdominal muscles but examination with the patient in a hot bath or under an anesthetic enables one to determine a swelling about the size of an egg in the right iliac fossa. It differs from the sausage-shaped swelling of an ileocolic intussusception. The bowels may move spontaneously or with cathartics. Blood in gross or minute quantities (by chemical test) is present in the stool during the cramp-like attacks. The remissions and chronicity differentiate it from acute invagination involving the entire gut.

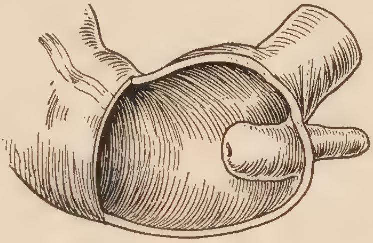

Fig. 312.—Intussusception of the appendix vermiformis (Cantlie).

**Obturation of the intestines** signifies the occlusion of the bowels by a foreign body situated therein; among such are gall-stones, enteroliths, foreign bodies which have been swallowed or, rarely, inserted into the rectum, lumbricoid worms, and masses of fecal matter.

Gall-stones, in exceptional cases if very large, may cause *an acute intestinal obstruction.* They probably have ulcerated through into the bowel from the gall-bladder. They may become impacted in the duodenum, jejunum, or at the *ileocecal valve.*

Cases of this last type are more frequent in women than in men. In many there is a previous history pointing to cholelithiasis. The symptoms are pain, violent vomiting (generally bilious), which frequently becomes feculent, constipation, which is not always absolute, meteorism, often not pronounced, and collapse. In some cases a hard resistant swelling may be felt. In others there will be some flatus from the rectum. Milder symptoms of occlusion may occur, evidently when the lumen of the bowel is not completely shut off. The patient in this class of cases would suffer from attacks of colicky pain, vomiting, and constipation, but no collapse. These cases may also have perforative peritonitis, and often die on the fifth to tenth day unless operated on. At times the stone may move onward and escape, with recovery of the patient.

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[1] N. Y. Med. Rec., Dec. 17, 1910.

Occasionally there will be some diarrhea with blood during the onward movement of the calculus, the result of traumatism to the mucosa.

*Enteroliths.*—An intestinal calculus rarely causes acute obstruction unless it has been dislodged from an intestinal diverticulum, and suddenly occludes the bowel. It generally produces the symptoms of chronic obstruction or digestive disturbances and obstinate constipation.

Calculi ordinarily consist of carbonates or phosphates with a foreign body as a nucleus; or as a result of the prolonged use of drugs, such as chalk, bismuth, magnesia or salol, or some indigestible material, such as oat-stones from oatmeal, etc., in which inorganic salts are deposited. They frequently develop in the large intestine.

*Foreign Bodies.*—These are accidentally or intentionally swallowed. This is especially apt to occur with children or with insane or hysteric persons. Occlusion has also been produced by the insertion of a foreign body into the rectum. Among such substances are knives, spoons, forks, keys, marbles, stones, false teeth, fruit stones, neckties, hair or beads, pins and needles; in fact, all varieties of objects. In some cases the symptoms may first be chronic, when suddenly acute occlusion will occur; in others it is acute from its incipiency. Murphy's button has caused occlusion, also a large mass of tapeworms or round worms. Pedunculated tumors of the intestinal wall, such as polypi, fibroma, and myoma, may produce it.

Many of these objects can be readily recognized by means of the *x*-rays and the fluoroscope, or by a Röntgen picture.

*Fecal masses* may completely occlude the intestines as a result of habitual constipation. The symptoms are usually subacute or chronic at first, and finally become acute from complete occlusion.

**Dynamic Ileus (Intestinal Paresis.)**—Acute obstruction produced by paralysis of the bowels is called paralytic or dynamic ileus. Among the various causes are hydrocele, contusion or inflammation of the testicles, etc., which may cause a reflex paresis; paracentesis of ascites; trauma over the abdomen; after laparotomy from manipulation of the viscera or following removal of a large tumor, or after Cesarean section; other operations from the effect of the anesthetic; damage to the intestines, as from a hernia after relief of the strangulation; local or general peritonitis; renal or biliary colic may cause reflex obstruction, possibly due to spasm or paresis; overdistention of the intestines from gas or feces; various toxemias, as from typhoid or pneumonia or from any acute infection, also sepsis, as from appendicitis, and as a concomitant of shock or uremia.

*Congenital Causes.*—Congenital occlusion of the rectum or colon produces acute obstruction.

*Acute Obstruction Engrafted on Chronic Obstruction.*—Chronic stenosis with chronic constipation may suddenly produce an acute obstruction.

**Pathology of Acute Obstruction.**—In acute obstruction the pathologic findings differ somewhat, according to the various types and factors. There has been an unfortunate tendency to confuse the findings in acute and chronic obstruction. If we have an acute condition engrafted on a chronic obstruction, we may then find above the point of obstruction dilatation and hypertrophy of the intestines, catarrh, and ulcerations.

In *true acute occlusion* the process is *too rapid* for these changes, but the following occur: the intestines below the occlusion are empty and contracted; above it the intestines are markedly dilated and distended and the walls are thinner. If a loop is involved, the walls are thick and congested (but not hypertrophied). The coils above the obstruction are distended, filled with gas and ill-smelling contents. If the small intestine is occluded the distention will involve the gastro-intestinal tract above the occlusion; if the large intestine is occluded the colon will be distended first; later regurgitation through the ileocecal valve occurs and universal distention.

In a loop which is strangulated there is local meteorism, and it may be markedly distended; there are congestion, edema, hemorrhage, a dark red color of the gut and gangrene, with general peritonitis and bloody fluid in the cavity; or a single perforation from ulceration or gangrene may occur (the distention ulcer with perforation "Dehnungsgeschwür," described by Kocher); and rarely, if the case be prolonged, a local abscess or local peritonitis.

**General Symptoms of Acute Obstruction.**—As a rule these begin very acutely, though rarely there is a previous history of diarrhea or constipation, improper food, a laxative, traumatism, or of violent exertion.

There are at first violent abdominal pains, colicky in type, local or diffuse. The pain is continuous, though sometimes it may remit. Nausea, hiccough, and vomiting first of the gastric contents, later bilious, and, finally, feculent vomiting rapidly follow. Meteorism quickly appears; there are absolute constipation and no passage of flatus. Tympanites increases, a great increase of intra-abdominal pressure is present, and the muscles become tense and the entire abdomen tympanitic; respiration is markedly interfered with, the breathing is rapid and shallow, pulse rapid and feeble, extremities cold, cold sweat, face pale, eyes sunken, and extreme thirst; total collapse and the patient rapidly succumbs.

Only a brief analysis will be made of these symptoms, as they have been thoroughly described under the respective causes of acute obstruction. Pain is the most constant symptom and it *never remits* completely. As a rule, it is more acute in obstruction of the small intestine; the initial pain is probably due to irritation of the intestines and peritoneum, later to spasmodic intestinal contraction, and finally to peritonitis. In the last stage pain may cease as a result of the terminal fatal intestinal paresis. External pressure, as a rule, increases the pain.

Active and visible *peristaltic movements* and tetanic stiffening of the bowel are *exceptionally* seen in acute occlusion, when the intestines were previously healthy and unobstructed; and if they are visible, are never as marked as in cases of acute obstruction *supervening on a chronic stenosis of the intestine.* This is an important point to remember. The visible peristaltic movements if present, in connection with the other symptoms, are an aid to diagnosis.

*Vomiting* is nearly always present. At first it is probably due to reflex irritation of the nervous system; later it has been ascribed to antiperistalsis; or to mechanical causes, as explained by Haguenot, who states that fluid contents accumulate above the obstruction; and from gas pressure and contraction of the abdominal muscles the contents are forced into the areas of less resistance and thus reach the stomach, where they cause vomiting.

Feculent vomiting often occurs when the obstruction is in the small intestine, due to putrefactive processes therein.

Constipation is usually marked; though rarely there is diarrhea with blood, as in intussusception.

Absence of flatus is quite significant.

*Meteorism.*—This may first be circumscribed (local), there being tympanites and protrusion of the intestines for a short distance above the point of obstruction; thus, a protrusion of the right side would first be noted if an obstruction occurs at the hepatic flexure. Later the meteorism becomes general and the *abdomen barrel shaped.*

*Collapse.*—This is marked, especially so if there is strangulation, and it is more rapid if the small intestine is involved.

Thirst and dryness of the tongue are present, being due to vomiting, sweating, and increased intestinal secretion, which diminish the fluidity of the blood.

Coma, delirium, and fever rarely occur.

**Diagnosis of Acute Intestinal Obstruction.**—Having described the symptoms of acute obstruction, a careful study of the physical signs and the methods of physical examination are necessary to complete the diagnosis.

Investigation of the type and degree of meteorism (tympanites), both by inspection and percussion, is of great importance. At this point I again desire to call to my reader's attention that acute dilatation of the stomach presents many symptoms of ileus and may obscure the diagnosis. The *pain* of acute ectasia is not as severe and continuous as with acute intestinal obstruction, in *that it is immediately relieved by lavage.* The stomach may occupy the entire abdominal cavity, though usually it fills the left half and lower part of the abdomen. The vomiting of acute ectasy is also peculiar, in that it is very profuse, incessant, and in large amount, and comes up in gulps without straining. It is usually watery and greenish in hue, though it may be brownish or black. If the stomach alone is involved, distention will disappear after lavage.

*Inspection.*—If there be occlusion of the jejunum or duodenum, unless the stomach also be greatly dilated, the upper part of the abdomen is protruded slightly.

Occlusion at the cecum or lower ileum gives the so-called "ladder" pattern, the coils lying one above another either obliquely or transversely in the abdomen, and the distention is more central.

In stenosis of the sigmoid flexure the upper and lateral aspects of the abdomen are usually distended, and we have so-called flank meteorism.

When the distention is limited to a section of the colon, flank meteorism may be unilateral; thus the right iliac region is intensely tympanitic if the occlusion involves the hepatic flexure. If the small intestine alone be included, the distended loops are seen in the center and flank meteorism is absent.

With volvulus the left lower portion of the abdomen protrudes; or an S-shaped protrusion of the abdomen, most prominent at the upper left and lower right quadrants, occurs.

We must remember that local meteorism occurs only early, and later general tympanites ensues, so that we may not see the case early enough to avail ourselves of these data.

Peristaltic waves, if present, aid to locate the obstruction.

*Palpation.*—In some cases palpation reveals a circumscribed area which is tender on pressure, and may aid in localizing the obstruction. In others a tumor is palpable, especially with intussusception, occlusion by tumors, or fecal impaction.

Palpation of the hernial openings is necessary. Digital examination of the rectum and vagina are most important. We may feel a stricture or intussusception in the rectum and bloody fluid may escape. I deprecate the method of inserting the hand in the rectum.

*Percussion.*—Local meteorism gives a deep and loud note, which is not truly tympanitic, but often of a metallic ring, and helps locate the obstruction. Exceptionally, dull percussion may be heard over the swelling, due to edema of the intestinal walls or to accumulation of fecal contents or blood admixture.

In normal subjects the percussion note in the upper lumbar region behind, is high, flat, and dull. It is loud and deep in stenosis of the large intestine. When the obstruction is in the sigmoid, the loud and deep note is found on both sides, and when in the splenic flexure or transverse colon, then only in the upper lumbar region of the right side.

If there is general tympanites and no change of percussion is noted for a long time over any one region of the abdomen, there is probably intestinal paresis. General tympanites, with absence or diminution of liver dulness, does not always mean perforation of the bowel, though it frequently does so. One can often determine peritoneal exudation by percussion.

*Auscultation.*—Splashing and gurgling noises often demonstrate that peristalsis is marked. Succussion sounds and fluctuation on palpation are frequently found in the intestines above the point of stenosis. Examination of the vomited matter will show whether fecal material is present. Urine is scanty and albuminous, and often shows indican and gives Rosenbach's reaction, especially in obstruction of the small intestine. Suppression of urine occurs in this condition. The writer opposes the use of $x$-rays for diagnosis in acute cases.

**Differential Diagnosis Between Obstruction of the Small and Large Intestine.**—*Obstruction of Small Intestine.*—Pain, vomiting, and collapse are more acute and appear early. Early feculent vomiting. *Indicanuria early, on second or third day. Anuria early, early meteorism, and often is high up.* Absence of early indicanuria (excluding peritonitis and intestinal inflammation) tends to show obstruction is not in the small intestine.

Vomiting in duodenal obstruction is rarely feculent, but is so if the obstruction is lower down.

*Obstruction of Large Intestine.*—If the disease is chronic or runs a long course, absence of indican shows lesion is present in the large intestine; its presence, however, tells nothing, as it may appear late.

General symptoms are usually less violent, though they are violent in volvulus. Fecal vomiting occurs later. Meteorism is lower down in the early stage of acute obstruction.

*Tenesmus* and *blood* in the stool suggest an obstruction low down. Brinton's method by the injection of water is sometimes of service. If not more than 1 quart can be injected, the obstruction is probably low down; if 2 quarts, it is probably above the sigmoid, and if 4 quarts, then in the commencement of the colon or higher up.

Inflating the bowel with $CO_2$ by Rose's bottle or by air will fill the large intestine up to the obstruction. This method is of no service if there is very marked distention, and lavage should be first performed to reduce it.

**Differential Diagnosis Between the Different Forms of Acute Obstruction.**—This is often difficult. In some cases it is possible, but in others we can only conjecture the probable type.

Acute strangulation in herniæ, omental slits, or by bands constitutes about 34 per cent. of cases. This is more frequent in males between twenty and forty years. In 90 per cent. the small intestine is the seat of trouble, mostly in the ileum and lower abdomen or right iliac fossa. The symptoms are sudden and acute. There may have been a previous history of peritonitis, hernia, or injury. Pains are severe, vomiting begins early and soon becomes feculent, absolute constipation is present and no passage of flatus. Tenesmus absent. Collapse is early and marked. Urine is scanty and meteorism slight. Attack is of fulminating type. Physical examination often gives no definite data.

*Volvulus.*—This most frequently involves the sigmoid. History frequently of chronic constipation, is more common in males and in those over forty years of age, from forty to sixty, though Fitz's statistics show frequency from thirty to forty years of age. If volvulus is of the small intestine, it cannot be differentiated from incarceration. Pain is sudden and violent, it may remit, but never intermits. Vomiting is quite common, but may be absent at first in volvulus of the sigmoid, and in the latter case fecal vomiting may not be present. It occurs in some cases, but is *not as frequent* as in internal strangulation; in fact, it is rather rare. Meteorism occurs early and is at first *local* in the *lower left quadrant,* or as an S-shaped protrusion. General meteorism rapidly occurs, and thus is an aid to diagnosis. The sigmoid can occasionally be felt *as a tumor.* Constipation and absence of flatus are usually complete; occasionally there is a little blood in the stool and only a moderate amount of water can be injected into the rectum. Symptoms are acute and collapse is marked.

*Intussusception.*—Most frequently in infancy and early childhood. Onset is sudden. Pains appear early, are colicky in character; in children, may cause them to scream or have convulsions; pains are paroxysmal. Vomiting occurs *early* and *constantly* in children; a less important symptom in adults.

Invaginated coil can be appreciated or a tumor in about one-half the cases. Occasionally the swelling may prolapse through the anus.

Tenesmus and *evacuation of blood* occur, then constipation and retention of flatus. Fecal vomiting rare, unless case is prolonged; collapse is early in children (in two to three days), but not as early as in strangulation; collapse is slower in adults.

*Obstruction by Gall-stones, Enteroliths, and Foreign Bodies.*—Occlusion from gall-stones occurs chiefly in older women. There is at times a previous history of gall-stones, jaundice, and liver enlargement or tenderness, which aids our diagnosis. The obstruction usually occurs in the small intestine. The general symptoms are often not so severe as in other types, there being some flatus passed at times and in some cases slight diarrhea with blood. If obstruction of the ileum, feculent vomiting is more marked. Meteorism is not so marked and collapse not so great. Occasionally the stone can be palpated.

*Enteroliths.*—Their recognition is quite difficult unless small fragments have been voided. They develop most frequently in the large intestine. Symptoms of chronic obstruction or digestive disturbances and obstinate constipation are more frequent. Acute symptoms are more rare.

*Foreign Bodies.*—The previous history and use of the $x$-rays will determine these. Obstruction from accumulation of fruit pits will generally be suspected by reason of the appearance of some of them in the stool.

*Ascaris Lumbricoides.*—An interesting case, demonstrating that round worms may be the cause of intestinal obstruction in a young child, has been reported by Charles Whelan.[1] The patient, male, age five and a half years, about April 1, 1910, became peevish, restless, complained of loss of appetite and frequently of stomach ache. The child became obstinately constipated. No worms were noted in the stools by the mother. On Aug. 2d, the child awoke about midnight crying with pain, and soon afterward began to vomit. This continued for two days, the abdomen becoming more and more distended. The vomiting increased in frequency and the pulse became extremely rapid, with evidences of shock. Several convulsions occurred, the child dying during a convulsive seizure. The autopsy showed an impacted mass obstructing the lower jejunum for about 6½ inches. The first 2 inches were completely occluded by a bunch of round worms, then there was a "kink" or constriction. Two worms lay side by side in the direction of the long axis of the bowel, passing through the constricted portion. The lower two inches of the jejunum were also completely occluded by the worms (Fig. 313). The condition was evidently an acute engrafted on a chronic obstruction.

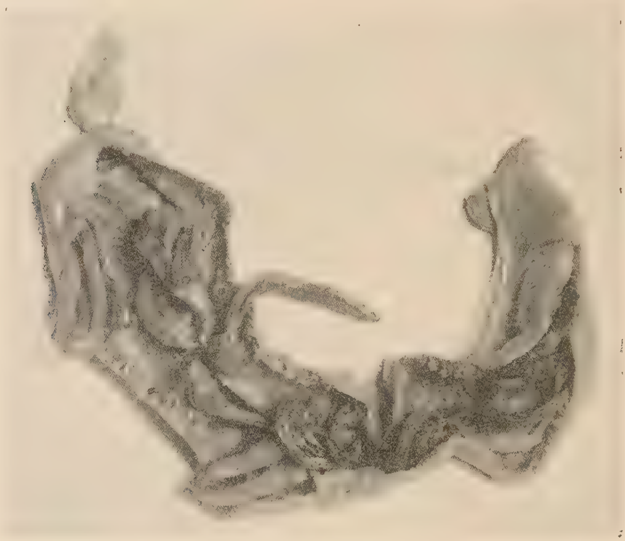

Fig. 313.—Section of obstructed intestine after opening into lumen, showing worms (Whelan).

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[1] Jour. Amer. Med. Assoc., Oct. 22, 1910.

*Fecal Accumulation.*—This rarely gives the picture of acute obstruction unless a tumor or stricture of the intestine be present, when accumulation may suddenly cause complete occlusion. Rectal examination usually shows hardened scybalæ, and palpation demonstrates a hardened mass in the colon (descending, sigmoid or cecum especially). If the mass be softer, it will impart a doughy feel on pressure.

*Dynamic Ileus.*—Acute gastroduodenal dilatation of the stomach presents symptoms of intestinal obstruction, and commences with acute pain, undoubtedly due to stenosis of the duodenum by pressure. Under Diagnosis of Acute Obstruction I have described the peculiar type of vomiting in acute ectasy and also the position of the stomach. The pain, distention, and other symptoms are relieved by frequent lavage and by the adoption of the abdominal position (the patient lying on his belly).

Obstruction of the bowel from a paralytic condition involves, as a rule, a considerable segment, or frequently the entire tract. After reduction of a hernia probably only a small segment is first involved, but I believe paralysis of a considerable segment, and in many cases of the entire bowel, finally results. The acute cramp-like pains, *persistent and paroxysmal*, are absent in this type of obstruction (really not a true obstruction at all), which always occur in the early stages of other types of obstruction. When acute spasmodic pain *is present and persistent*, I always feel positive of a true obstruction. The vomiting, constipation, collapse, etc. may be similar to obstructive ileus. There may be sudden pain with the acute distention, so that perforation is suspected, but after lavage and enteroclysis the pain disappears, and muscular rigidity is found to be absent. These features exclude, first, "obstructive ileus," on account of disappearance of the pain; and second, "peritonitis." In the early stages of "dynamic ileus," distention, interference with the cardiorespiratory functions, and obstinate constipation precede the vomiting and severe symptoms as a rule. There is usually more of a sense of painful discomfort and oppression, except in the cases of sudden acute distention.

In the terminal stages of true obstruction, paresis of the bowel may occur, and then *spasmodic pain disappears;* in paresis of the bowel from peritonitis there is the general pain and tenderness from the peritonitis, but not the acute spasmodic pain peculiar to true obstruction.

In true dynamic ileus, therefore, there is the absence of that acute *persistent spasmodic* pain of severe type which is present in all cases of true obstruction.

**Differential Diagnosis Between Intestinal Obstruction and Other Diseases.**—*Acute Peritonitis.*—Generally a history of local peritonitis, such as appendicitis with pain localizing in the right iliac fossa, and then sudden cessation of the cramp-like pains, followed by abdominal tenderness, muscular rigidity, great distention, and frequently a rise of temperature. Vomiting generally begins later and feculent vomiting still later, etc.

In obstruction the character of pain is more severe and persistent, as a rule, and often in the umbilical region or in the left lower quadrant; temperature is subnormal at the start; abdominal tenderness is not as marked; feculent vomiting earlier; often local meteorism before general tympanites. General pains are colicky and persistent until peritonitis and paresis set in. The *paralytic form of ileus* may often occur with infectious diseases, or acute appendicitis with peritonitis (general), also after operation or inflammation in the pelvis or genito-urinary organs; and the knowledge of these facts aid our diagnosis. We must remember that in hysteric women all the symptoms of ileus, even to fecal vomiting, may occur without there being any obstruction.

With biliary and renal colic there may be a reflex paralytic ileus, but the symptoms of these conditions aid the diagnosis.

Lead- and arsenic-poisoning have sometimes been mistaken for ileus, but again we have the history and other symptoms, especially of enteritis. Simple intestinal colic soon subsides under treatment. Acute pancreatitis and enteritis give their symptoms.

There is tenderness in the course of the pancreas if this is involved, a circumscribed epigastric swelling, and tender spots throughout the abdomen (Fitz); but in some cases it is difficult to differentiate acute pancreatitis from obstruction. The former presents the aspects of an *epigastric peritonitis*. Fat necrosis is found on operation.

**Course.**—The course of acute obstruction depends upon its cause and site. The higher up in the intestine, the more acute the course, as a rule.

Volvulus and strangulation are very acute. The patient may die in collapse within a few hours, or the course may be prolonged to two or three days or even a week. Intussusception, if unoperated, may last several weeks. If the patient survives the collapse, and the patency of the gut becomes reëstablished (as in intussusception, foreign bodies, or volvulus), flatus is first passed and then later a fecal movement, and the symptoms gradually abate. The invaginated bowel has been known to slough off and be passed in the stool. From changes in the gut, due to ulcers, adhesions, etc., the patient may subsequently develop symptoms of chronic obstruction.

If operation is not performed, the patient usually dies of acute shock or peritonitis. The latter may be due to perforation or to direct penetration of the paralyzed wall of the gut by intestinal bacteria. It is further believed that toxemia from intestinal bacteria may be a cause of death.

In some there may be a circumscribed peritonitis. With the diffuse peritonitis we have the symptoms already described.

Embolic processes may develop in the liver, lungs, and other organs. There may be aspiration pneumonia, or exceptionally, local abscess, with perforation of the abdominal wall, or into another part of the intestines or into other viscera, as into the stomach, bladder, vagina, or uterus.

**Prognosis.**—The prognosis of acute obstruction is very serious. Some observers state that about one-third of all cases recover, however.

Obstruction caused by coprostasis, gall-stones, or some foreign body give the best prognosis. Dynamic ileus with modern methods of treatment, I believe, is next in regard to favorable results, then intussusception, and the worst cases are volvulus and strangulation. The *earlier the operation, the better the prognosis.* The last types I believe fatal nearly invariably, unless early operation is carried out.

**Treatment.**—This may be divided into medical and surgical, and the respective indications are extremely clear. I shall first briefly classify these, giving the treatment in tabulated form.

*Cases for Medical Treatment.*—1. Acute obstruction due to fecal accumulation. Acute attacks are rare; they are, rather, *subacute* or acute engrafted on chronic:

(*a*) Lavage is indicated to relieve tympanites, if present, or if vomiting.

(*b*) Digital examination of the rectum and removal of scybalæ with the fingers, and then frequent enemata of soapsuds, olive oil, glycerin, or 6 ounces (200 c.c.) of magnesium sulphate (saturated solution) in water, 1 pint (500 c.c.), followed by recurrent enteroclysis with normal saline solution at 110° to 120° F., using 2 to 3 gallons at a sitting, about $\frac{1}{2}$ to 1 pint (250–500 c.c.) being kept in the bowel. For the first twenty-four to forty-eight hours the treatment consists in the simple mechanical emptying of the rectum and large intestine. The enemata should contain about 1 to 2 quarts (liters), in which is olive oil, 1 pint (500 c.c.), alone or with glycerin, 4 ounces (125 c.c.). It is well to give the enema with the patient in the knee-chest position, the buttocks elevated as high as possible. In two recent cases the writer found a high enema containing olive oil, 8 ounces (250 c.c.); glycerin, 4 ounces (125 c.c.); castor oil, 2 ounces (60 c.c.); and several grains of ox-gall, given with a large *hand syringe* under considerable pressure, to be of great value. The soft rectal tube was inserted as high as possible. If vomiting or distention, *no food at all until this ceases;* thirst may be relieved by moistening the mouth, sucking a piece of lemon or orange, and, if necessary, by hypodermoclysis, rectal saline enemata, or proctoclysis. Milk is objectionable, as it forms curds and helps fill the bowels. Broths and soups should be given until the accumulation has been removed. This only refers to after the *subsidence of acute symptoms* and after the *bowels have acted.* No food is given before this time. Strychnin by hypodermic, $\frac{1}{60}$ to $\frac{1}{30}$ grain (0.00108–0.0021) three or four times a day, can be given as a stimulant. It also helps tone up the bowel.

Tincture of belladonna in large doses, administered up to physiologic symptoms, 10 drops (0.59) three or four times a day, is of service, as soon as it can be retained. Later, olive oil, 2 to 4 ounces (60.0–125.0), can be given several times a day by mouth. On the second or third day, if matters are progressing favorably, give cathartics, calomel, castor oil, etc., by mouth. Massage and external electricity are of value in these cases after the acute symptoms have subsided. I have frequently employed electric enteroclysis. I have seen it take several weeks to completely empty the bowel.

2. The second class of cases in which medical treatment is indicated is in dynamic ileus. The indications are:

(*a*) Abolition of food.

(*b*) Relief of thirst, as previously indicated.

(*c*) Frequent lavage to relieve intra-abdominal tension and also vomiting.

(*d*) Frequent enteroclysis (recurrent) with hot normal saline solution at 115° to 120° F., several gallons being used, a small part being kept in the intestines (preferably). High enemata with soapsuds, olive oil, and glycerin are also of service.

(*e*) Electric enteroclysis.

(*f*) Tincture of belladonna, 10 drops (0.59), given three or four times a day up to physiologic symptoms. Strychnin, $\frac{1}{30}$ grain (0.002), every four hours.

(*g*) Magnesium sulphate (saturated solution) by rectum, 4 ounces (125.0). Heat locally is of value, and in one case the continuous local application of the ice-bag stimulated the bowels to contract. I prefer the ice-bag in many cases, unless collapse is present.

Cathartics by mouth immediately after lavage, especially calomel, 5 grains (0.3), crushed up in water and poured in through the stomach-tube at the end of the washing. Plain water and *no saline solution* should be employed for lavage when *calomel is administered*. Physostigmin sulphate (eserin), $\frac{1}{100}$ to $\frac{1}{50}$ grain (0.0006–0.001), every two to three hours for three doses. It should be arranged to give strychnin, $\frac{1}{60}$ grain, with each dose. Some recommend elaterin, $\frac{1}{10}$ grain. If all methods fail and the patient is rapidly losing ground, then simple enterostomy of the most distended loop, under cocain, as suggested by Elsberg in obstructive ileus, I believe, is indicated. This procedure allows the escape of gas and some contraction of the intestines to take place. Magnesium sulphate solution should be injected through the opening, and the procedures already suggested should be continued. Drainage should be closed after twenty-four hours. In my own experience I have had *good results from continuous and active medical treatment*. The *lavage* followed by catharsis by mouth is of *equal importance* as the *enteroclysis*. Proctoclysis is not sufficiently active.

*Obstruction by Foreign Bodies.*—If the obstruction is by accumulation of fruit stones, an enterolith, or a gall-stone, and the case is seen on the first day of attack, lavage and the administration of 4 ounces (125 c.c.) of olive oil through the stomach-tube aids to lubricate its passage, followed by enteroclysis. *No cathartic* should be given by mouth. If the mass begins to come away and symptoms are relieved at once, then delay; otherwise, operate.

If the case is seen later, with progressive symptoms, use lavage and enteroclysis to relieve distention and operate at once.

*Intussusception.*—With infants or young children the stomach should first be washed out to relieve abdominal tension. An anesthetic is then administered and the child placed in the Trendelenburg position, and an attempt at reduction may then be made by inflation. A bellows is attached to a catheter, and the air should be injected slowly, the buttocks being held together. The best guide to the amount introduced is the tension of the abdominal walls; if tension is marked some air is allowed to escape.

This procedure should not occupy over fifteen to twenty minutes.

A saline solution, milk and water, or thin gruel at a temperature of 100° to 105° F., for the relaxing effect can be employed instead (as suggested by Holt). The fluid is suspended in a fountain syringe 4 or 5 feet above the patient's head, the tension of the abdomen being watched. Otherwise the procedure is the same.

Reduction is indicated by a rumbling sound and by the abdomen resuming its natural contour, with the disappearance of the tumor; in some cases a gush of feces follows.

If these symptoms are absent, the abdomen is examined while the patient is still under chloroform, especially the right iliac fossa, for the continued presence of the tumor.

It is better not to repeat the injection.

If the tumor is present, or if vomiting continues and no gas or feces are expelled, or the pulse and temperature rise, immediate operation is indicated.

This method of taxis should be tried on the young in an early stage (the first day); if later, operate. If, on the other hand, there is immediate improvement, small doses of opium are given for a few days to prevent re-invagination.

*Surgery.*—In all other cases of acute intestinal obstruction except those noted, early, *preferably immediate, operation* is indicated.

A large percentage of fatalities imputed to operation are due to delay on the part of the physician; thus, in strangulation, volvulus, intussusception, and obstruction from foreign bodies (except under the condition of the rapid passage of foreign bodies, as noted above), immediate operation is indicated. *The earlier the operation* the better the prognosis.

In these cases *cathartics should never be given*, the bowel should not be massaged, nor electricity used.

*Puncture of the bowel through the abdominal wall to relieve gas is absolutely dangerous*—an invitation to peritonitis.

*Laxatives do positive harm.* The attempted diagnosis of the seat of the acute obstruction by the administration of bismuth by mouth and the use of *x*-rays, which necessitate a *delay of six hours or more*, should never be undertaken. Bismuth enema, followed by the *x*-rays, is only of use in determining obstruction in the large intestine. It is of value in chronic cases, but not advisable in acute ones.

Lavage (frequent) is of first importance; it lessens intra-abdominal tension and also the pain, makes diagnosis easier, and in some cases has actually proved curative. This last is explained by the fact that the gastro-intestinal distention is relieved above the point of obstruction, and occasionally the gut escapes from the constriction.

If the patient is seen on the first day of the attack, lavage should be given at least twice, one or two hours apart, while preparing for operation; if later, then once before operation.

Enteroclysis at 115° to 120° F. is of value to lessen the distention due to general paresis and improve the pulse; if the intussusception lies in the rectum, irrigation is contra-indicated.

No fluid or ice by mouth, but the tongue can be moistened and small normal saline injections or proctoclysis be administered for thirst. These methods also relieve postoperative thirst. Heat or cold to abdomen. Hypodermoclysis and infusion if there is shock.

Lavage often relieves the pain, but if this is very severe, morphin, $\frac{1}{4}$ grain (0.016), by hypodermic, and repeat to lessen shock. All these methods are of use while preparing for immediate operation.

The general medical methods of treatment by opium or morphin for three or four days and expectant treatment, with *subsequent operative* fatality, *are to be deplored.*

Operate in acute peritonitis. Subsequent to operation, lavage, if vomiting, and nutritive enemata; no food or water by mouth; later, a little hot water. Proctoclysis is of value for the sepsis. Open bowels by enema or enteroclysis in twenty-four to forty-eight hours, as intestinal paresis is usually present; earlier if the *symptoms persist* which are due to this condition.

Rectal injection of 400 c.c. (12 ounces) of 7 to 8 per cent. salt solution in intussusception to produce reversed peristalsis has been suggested by Riegel. Experimentally it has proved efficacious, but practically I am dubious of its value, as most cases of intussusception are adherent or strangulated.

In desperate cases enterostomy with drainage of the intestines, done under local anesthesia (cocain), is advisable. A few hours later (within twenty-four hours) radical operation with relief of the obstruction can be performed. Elsberg[1] holds that preliminary enterostomy, leaving the prolonged search for the obstruction for a second operation, to be more frequently advisable.

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[1] The Value of Enterostomy and Conservative Operative Methods in the Surgical Treatment of Acute Intestinal Obstruction, Ann. Surg., May, 1908.

## CHRONIC INTESTINAL OBSTRUCTION

In this condition there is a stenosis or narrowing of the lumen of the intestines, but the obstruction is not acute and complete in the earlier stages, but comes on gradually.

**Etiology.**—It may be caused by the same factors which produce acute obstruction, if the entire lumen of the canal is not occluded.

One of the most frequent causes of chronic obstruction is stricture resulting from ulcers or new growths. The latter, even if they do not occupy the entire canal, may protrude at one point and partially occlude the intestines. They may be benign or malignant.

In addition we have the peculiar tumor-like tuberculosis of the cecum, which causes a progressive stenosis, and chronic peridiverticulitis (sigmoiditis) may produce a narrowing of the lumen with symptoms. Both of the latter conditions often simulate carcinoma.

Strictures resulting from ulcers involve the large intestine much more frequently than the small, probably in a ratio of 6 to 1, according to Treves.

Among the causes of stricture are tubercular, stercoral, syphilitic, typhoid, dysenteric, and duodenal ulceration.

Woodward[1] has demonstrated that dysenteric ulcers rarely cause intestinal stricture, and Nothnagel agrees with him. Stercoral and tubercular ulcers are a quite frequent cause, as is also syphilitic ulceration. Typhoid ulcers are a rare cause.

There are other rare factors reported, such as ulceration in a portion of the bowel that has been incarcerated, or stricture following the sloughing off of invaginated intestines. A few cases of traumatism with damage to the intestines and subsequent stricture, or of a circumscribed peritonitis with adhesions, following trauma, and subsequent stenosis have been reported. Carcinoma of the pancreas, or chronic pancreatitis with enlargement of the head of the pancreas, enlargement of the retroperitoneal glands, and gall-stones may cause stricture. A stricture may be congenital.

Rectal strictures are quite frequent, and much more so in women, as from syphilitic, tubercular, stercoral, and hemorrhoidal ulcers. Operation for prolapse of rectum or for hemorrhoids, especially the Whitehead operation, traumatism from the syringe-tip, or the introduction of foreign bodies may produce stricture.

Traumatism from the child's head during parturition may be a cause.

Gonorrheal abscess of the Bartholin glands may lead to ulceration of the rectum and stricture ultimately results.

Adhesions may be a frequent cause of chronic stenosis, sometimes of rather a mild type with persistent constipation of long duration as the chief symptom, but no actual obstruction. The

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[1] Medical and Surgical History of the War of the Rebellion.

Lane kink due to adhesions of the ileocecal junction, various *angulations* in extreme enteroptosis not relieved by medical treatment and prolapse of the sigmoid may be causes. In a recent case of T. A. Gonzales, seen by the writer in consultation, a point of stenosis was found at the sigmoidorectal junction due to adhesions, but more marked and unsuspected adhesions were demonstrated by S. Tousey by a röntgenograph. The case was successfully operated on by Parker Sims, confirming the $x$-ray diagnosis.

**Anatomy of Chronic Stenosis of the Intestines.**—The intestines below the stenotic area are empty and contracted and the bowel normal. Above the point of stenosis the bowel is dilated, often to a great degree, and may form a sac-like pouch. The dilatation may involve only a short part of the bowel above the stenosis.

In some cases there is considerable distention from gas and the abdomen may assume *the barrel shape*. The degree of distention depends on the tightness of the stricture. There is stagnation of the intestinal contents above the stricture, which causes mechanical distention and may stimulate the peristaltic action. This material also acts as an irritant. When the musculature is stimulated to increased activity *hypertrophy* is thus produced. This accounts for the *violent visible peristaltic movements* in chronic obstruction.

Patel claims that in stenosis from external pressure we have dilatation without hypertrophy, and that the latter only occurs if ulcers, which cause contraction through irritation, are present. This would explain the occurrence of hypertrophy without stenosis in some cases of intestinal ulceration.

Changes in the mucosa and submucosa are frequently present, a catarrhal condition and ulceration (stercoral). General peritonitis, local peritonitis, or local abscess may result.

The intestines may become elongated above the stenosis.

*Location of Stenosis.*—The large intestine is the most common seat of the stricture. Syphilitic, dysenteric, and stercoral strictures are chiefly localized here, as are also those resulting from traumatism or from follicular ulceration.

Tuberculous ulceration produces stricture most commonly in the small intestine, though tuberculous tumor is found in the cecum, and probably tuberculous ulcer is more frequent in the rectum than has been usually credited. Tuberculosis of the sigmoid also occurs.

Malignant strictures are most frequent in the *large intestine.*

If the ulcer producing the stricture lies parallel to the longitudinal axis of the bowel, stenosis is not as marked as when it is an annular ulcer (girdle ulcer). The stricture, as a rule, is short and the external aspect of the intestine looks as if a ribbon had been tied about it. The external surface of the gut is often covered with exudate, so that the bowel is thickened and there may be adhesions between the intestines and other loops or other viscera, which further constrict the intestines.

The stricture consists of cicatricial tissue unless malignant. Folds of mucous membrane near the cicatrix or hypertrophic polypoid protrusion of mucous membrane may aid in closing the gut.

If there are numerous stenoses, as from tubercular ulcers, there may be sacculated dilatation of the small intestine between the stenosed points.

**Symptoms.**—These depend upon the cause of the obstruction; malignant growth must be differentiated clinically from benign conditions. The symptoms generally come on gradually.

In stenosis of the small intestine they may be latent for a considerable time, on account of the fluidity of the bowel contents, and then appear with rapidity. On the other hand, stenosis of the colon produces symptoms more rapidly on account of the solid contents.

As a rule, *constipation* is one of the earliest symptoms, and this gradually becomes worse. The patient complains of digestive disturbances and swelling of the abdomen, there are loss of appetite, and nausea. Stenotic feces in round balls, cylinder (pipe-stem) or tape-like movements, are suggestive, but not conclusive. This type of feces may occur in spastic constipation, and, on the other hand, the feces may be normal in form with a stricture high up. Diarrhea at times alternates with constipation. The diarrheal movements may be extremely offensive and contain mucus, or even pus and blood; *this last is especially true in malignant stenosis*, where there are active ulcerations, or in intussusception. The diarrhea often relieves the patient. We must remember that chronic diarrhea is present in some cases when there is a *marked catarrh* above the stricture, and this will sometimes lead one astray. *Severe pain* of a colicky type occurs in all cases, and this may at times be excruciating; it may be localized near the seat of stricture, but in other cases be more diffuse, radiating even toward the thorax and producing a feeling of oppression and dyspnea. A symptom which occurs with the colic, which can be considered pathognomonic, is the visible *peristaltic movement of the intestines*, in which the loops can be seen to stiffen and relax alternately. The coils appear and disappear. Peristaltic unrest (intestinal) is not always present, especially in the earlier stages of the disease; and at times, late in the condition, the bowel may become fatigued and paretic from overdistention. Often, however, the peristaltic movements will aid in locating the position of the obstruction.

Vomiting may not occur at first, but is later more frequent, and if finally the obstruction becomes complete, may be marked and even feculent. Gurgling, rolling sounds, and meteorism are present.

**Location of the Obstruction.**—*This influences the character of the symptoms.* If the stenosis is situated above Vater's papilla, the symptoms are similar to those of stenosis of the pylorus. Marked dilatation of the stomach, nausea, and vomiting are prominent. R. T. Morris has demonstrated that in some cases of spider adhesions from the gall-bladder there may be severe hemorrhage and pain which may simulate gastric ulcer. There is usually a history of previous gall-bladder disease. If the obstruction lies below Vater's papilla, we again have gastric dilatation, but frequent bilious vomiting. The stomach-contents are *neutral* or *alkaline*, due to regurgitation of pancreatic juice and bile, and duodenal digestion takes place within the stomach. With stenosis above the papilla, gastric contents, as in benign pyloric stenosis, are acid. Riegel states another sign of duodenal stenosis is, that when the stomach has been emptied the night before, twelve hours later as much as 3 liters (quarts) can be frequently aspirated. The reaction and contents differ from that of gastrosuccorrhea.

The lower down the obstruction, the less pronounced the gastric symptoms, as a rule, and the more marked the constipation and colicky pains. There may even be an absence of gastric symptoms for a considerable period if the stenosis be in the lower ileum or in the colon. The symptoms, however, are always more severe in chronic intussusception or chronic types of strangulation than in ordinary cicatricial stenosis.

**$x$-Rays.**—The administration of bismuth by mouth and inspection the following day with the fluoroscope or by photography may demonstrate the location of the stenosis. If it be apparently in the large intestine a high injection of bismuth and olive oil or bismuth suspended in water is given, and the examination is then made. These procedures are not to be recommended in acute cases, though valuable in the *chronic for diagnosis.*

**Inspection of the Abdomen.**—When the stenosis is high up in the small intestine there is apt to be distention of the epigastric region, and when in the lower part of the small intestine or in the large intestine, then there is considerable abdominal distention.

The active peristaltic contraction of the intestines is marked, the coils (stiffened) rising and falling, and often performing winding or vermicular motions. These movements are associated with colicky pains and with gurgling and rolling noises.

Sausage-shaped ridges may appear with depressions in their vicinity, and in a few seconds the ridges disappear in one part and appear in another, the coils never remaining visible in one place for any length of time. They appear hard and stiff to the hand and then suddenly become elastic.

The contractions in the small intestine are usually smaller than in the large intestine.

Meteorism in the milder cases is not marked, as the gas can pass the obstruction. Later it may become quite marked, and, as in acute obstruction, may be local or general in character.

If the obstruction is in the lower colon or rectum, it will be most pronounced at first in the course of the colon, on the sides of the abdomen, and in the epigastrium.

If in the lower ileum, the distention is more pronounced in the umbilical and hypogastric regions, and the lumbar regions are relaxed (undistended).

Later there may be more general distention and the *barrel-shaped* abdomen. Local manifestations of meteorism are described under Acute Obstruction.

One of the important types is chronic intussusception, which may develop after an acute attack has subsided or may occur as such from its incipiency. It takes place most frequently in the ileocecal form. Some of the latter cases may continue for months or even years. The pain is paroxysmal, but may entirely intermit. There may be attacks of pain daily, or every few days or weeks. As the disease advances the intervals grow shorter. Vomiting is not marked. Often diarrhea is present, or constipation alternating with diarrhea. Blood may be passed and tenesmus is at times present. Palpation shows the presence of a tumor in about 50 per cent. of the cases, or a tumor can be felt in the rectum. Local meteorism may be present. Occasionally the invaginated part may slough off and perfect recovery ensue or, again, ultimate stenosis may follow. Death may occur through perforation.

**Chronic Obstruction through Fecal Accumulation.**—This is more common in females and usually in our older patients. The history is of habitual constipation. At times large amounts of fecal matter are voided by enema. Scybalæ are frequently present. There are digestive disturbances, flatulence, loss of appetite, eructation, fetid breath, headache, dizziness, and symptoms of autointoxication; there may be oppression in breathing from distention, an unhealthy appearance of the skin, and a foul tongue. There may be pains in the thighs, legs, and genitals due to pressure on the lumbar or sacral nerves. The patient may be very neurasthenic.

Distention and gurgling may occur. There are colicky pains, but usually they are not severe.

If untreated, the condition may become worse, the constipation increase, and all the symptoms of chronic or even of acute obstruction develop, as already described under Fecal Obstruction. The vomiting may even become feculent.

Palpation shows the presence of a tumor frequently in the colon, especially in the cecum, sigmoid, or other flexures. It may be hard and uneven, and will often " pit " on pressure. It is not painful on pressure, as a rule. Rectal examination generally shows the presence of scybalæ. I have treated a case in which apparently nearly the entire abdomen was occupied by the fecal tumor. Operation for " tumor " had been advised. As a result of 80 bowel movements in one week the tumor disappeared. The patient had had no bowel action for three weeks. Enemata, and especially recurrent rectal irrigation, and later, catharsis will reduce the size of the tumor. I have seen cases in which acute flexions or angulations of the sigmoid and colon have been factors in the production of this condition. They are well described by J. P. Tuttle.[1] Gant has also demonstrated that prolapse of the sigmoid flexure is a cause, and relief has been secured by suspension of the sigmond.

**Rectal Stricture.**—There are the symptoms of progressive constipation at times with alternating diarrhea, with mucus, and at times, pus with traces of blood in the stools, colicky pains, tympanites, tenesmus, and loss of appetite. There may be hemorrhoids and rectal prolapse. Digital examination, exploration with a rectal bougie or the proctoscope, will demonstrate the constriction.

With stricture the pressure on the examining finger remains constant and is not like sphincteric spasm, which soon relaxes. Many of the strictures are within the finger reach, within 4 to 6 centimeters up the bowel; if not within reach but suspected, a Wales rectal bougie or soft tube can be employed for the examination. The degree of stricturing can be determined by using tubes or bougies of varying sizes. It is preferable to pass the speculum up to the point of stricture, so as to examine its nature thoroughly. It is often advisable to remove a small section under cocain for microscopic examination. With malignant stricture there are cachexia, loss of weight, metastases, and the symptoms described under Carcinoma.

**Complications.**—Above the stenosis on account of the ulceration we may have circumscribed peritonitis and abscess or perforation with general peritonitis. The abscess may rupture into other viscera or perforate the abdominal wall. The chronic condition may suddenly become acute, and severe collapse occur with death, or thrombosis of some of the veins, or pyemic processes, or the patient may die of inanition.

**Diagnosis.**—The gradually increasing constipation; colic attacks with frequent stoppage of the bowels, alternating at times with diarrhea and the temporary relief of symptoms; visible peristaltic movements with tetanic rigidity of the intestines; at first local meteorism and, later, the tendency to the barrel-shaped abdomen; the presence of gastro-intestinal disturbances of varying degree and, frequently, loss of weight are suggestive of the obstruction. Rectal examination is always of importance.

If the constipation is of *long standing*, in an elderly person or invalid, and there is no cachexia, the tumor movable and doughy on pressure, scybalæ being passed, and at times accumulation felt in the rectum, fecal tumor is evident. These occur chiefly in the caput coli, sigmoid, colon flexures, or rectum.

With carcinoma there is marked cachexia and the tumor is hard and solid, occurs mostly in the caput coli, sigmoid, and rectum, and more frequently in persons over forty-five. There is slight or moderate leukocytosis, and also anemia. In the scirrhous type of carcinomatous stricture, cachexia may be scarcely noticeable for a considerable period. The progressive constipation and age of the patient are significant together with increasing anemia.

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[1] N Y. Med. Jour., etc., March 14, 1908.

Tuberculous tumor of the cecum and peridiverticulitis (chronic) of the sigmoid must be held in consideration as causes.

With chronic intussusception the mass is usually of sausage shape, and shows the peculiarity that it sometimes feels hard and sometimes soft on palpation; there are mucus and blood in the stool.

External tumors can generally be appreciated. Vaginal examination should be made. If there have been attacks of peritonitis, bands and adhesions would be suspected.

A previous history of diarrhea, dysentery, syphilis, or tubercular difficulty would suggest ulcerative stenosis.

Stricture of the small intestine is most frequently due to adhesions or tubercular ulcer. In the large intestine stercoral, syphilitic, or dysenteric ulcers are to be considered, chiefly carcinoma or pelvic inflammation, perityphlitic adhesions, and, more rarely, tuberculosis of the cecum and chronic peridiverticulitis.

**Course and Prognosis.**—This depends on the etiology and severity of the obstruction. With non-malignant stricture of the bowel of moderate type, or in the fecal obstruction cases, not progressive, the patient by proper regulation of diet may live many years. With malignancy the prognosis is fatal unless relieved by early operation; chronic intussusception occasionally clears up, but the prognosis, as a rule, is bad. In progressive cases the symptoms rapidly become worse and life is shortened, with death from final acute obstruction. Unless radical operation is performed, complications such as peritonitis, pyemic processes, etc., hasten the final result.

**Treatment.**—If chronic obstruction suddenly or gradually develops into acute obstruction, the same indications for treatment exist as in the latter condition.

In the chronic cases the proper regulation of diet, omitting those things which will mechanically fill up the intestines and a careful regulation of the bowels, are most important. Substances that give a large residue of fecal matter, which are irritating and extremely constipating, should be excluded.

Matzoon, koumiss, buttermilk, bacillac, lactone-buttermilk, and kefir milk are excellent. Milk agrees well, as a rule, with most cases and is readily digested, while others it constipates and is undigested.

Raw eggs can be beaten up in milk, and soft-boiled eggs, broths, soups, and gruels administered. Sanatogen (flavored), tropon, and somatose are of value, administered in the broths or milk.

If the stenotic symptoms are progressive or fairly marked, liquid or soft diet alone should be given.

In milder cases scraped beef, tender meat (well divided), butter, a small amount of well-toasted bread, and moderate in quantity, rice, sago, and mashed potatoes in small amount are admissible.

The patient should eat a small quantity frequently and should take sufficient food to preserve his nutrition. Very hot and cold drinks should be avoided. Irritating food, such as mustard, spices, pepper, vinegar, fruits in bulk, and green vegetables in large amounts, should be forbidden. Substances giving a large residue of fecal matter should also be cut off. Spinach I have found of service to aid bowel action. Fats, such as cream and butter, are useful.

Fresh fruit juices are valuable, and the administration of a glass of water on rising is of service.

*Bowels.*—The bowels must be moved every day. Injections of soapsuds enemata of medium size, not over 1 quart (liter), with the hips elevated; Kussmaul's method of oil injection, 1 pint (500 c.c.) or more, being retained over night; or the addition of olive oil to the enema, mild cathartics, such as cascara, rhubarb, syrup of figs, phenolphthalein, regulin, and occasionally sulphate of magnesia or Apenta water, are of service. Under Chronic Constipation numerous remedies are described.

Enteroclysis (recurrent) is valuable in many cases.

Massage, vibratory massage, and electricity are of service only in *cases due to fecal accumulation.*

If diarrhea is present, unless the patient is weakened thereby, it should not be checked. In the latter event mild preparations, such as bismuth subnitrate, chalk mixture, or chalk and catechu, are preferable to opiates. If there are small diarrheal movements, evidently a diarrhea associated with constipation, then a dose of castor oil, laxol, or a saline cathartic is indicated.

*For Colic Attack and Peristaltic Movements.*—Hot applications to the abdomen are indicated and a recurrent irrigation of normal saline solution at 115° to 120° F. is of value, to be given for ten to twenty minutes. The latter removes gas and clears the bowels. Enemata also can be administered.

Tincture of belladonna, 10 minims (0.59), once or twice, is of use to allay spasm, or by suppository, extract of belladonna, $\frac{1}{4}$ to $\frac{1}{3}$ grain (0.016–0.024). Opium I avoid if possible, and then only $\frac{1}{8}$ to $\frac{1}{4}$ grain (0.008–0.016) of morphin or codein by hypodermic. It should be given only to allay severe pain.

For vomiting and distention lavage is indicated.

Cerium oxalate with bismuth and soda will check vomiting, also 1 minim (0.059) doses of Fowler's solution of arsenic every hour for four doses.

Cocain, which has been suggested, is a dangerous and pernicious drug. I have seen complete collapse after its use in as small doses as $\frac{1}{10}$ grain (0.006).

Lavage is of special value in temporarily allaying symptoms if the stenosis is in the small intestine.

If there is chronic fecal impaction, our first efforts should always be directed from below.

Hardened scybalæ should be removed from the rectum by the finger, oil injections, and, later, by soapsuds enemata and recurrent rectal irrigations. Olive oil can be given by mouth.

Later, cathartics, massage, electricity, and vibratory massage are of service.

Electric enteroclysis I have found useful in obstinate cases of fecal impaction.

Tincture of belladonna, 10 minims (0.59) three or four times a day, and strychnin, $\frac{1}{30}$ grain (0.002) t. i. d., are of value. Eserin sulphate, $\frac{1}{100}$ to $\frac{1}{60}$ grain (0.00065–0.00108), may be required.

Mild cases of rectal stricture, *providing they are not malignant*, can be benefited by dilatation for ten to fifteen minutes every two to three days with different sized bougies. Operation on the stricture may be necessary in some cases. Thiosinamin can first be tried, injected into the bowel or by hypodermic. Dose, $\frac{1}{2}$ to $1\frac{1}{2}$ grains (0.033–0.1); or by hypodermic, 1 grain (0.065), in 15 per cent. alcohol solution or 10 per cent. glycerinated solution.

*Operation.*—Most types of chronic intestinal obstruction grow worse, except those due to fecal impaction. In mild types of rectal stricture local dilatation may be palliative and keep the patient comfortable.

Malignant growths must be extirpated as soon as possible. Strictures must be treated according to their location—in the rectum, by division and dilatation or by resection. In other regions, enteroplasty, splitting the gut parallel to its axis, at the same time dividing the stricture and uniting the incision transversely, has been successful. Complete excision may be necessary, with or without employing Murphy's button to unite the ends. Anastomosis of the bowel above to that portion lying below the stricture may be required. In stricture of the colon in cases *quite prostrated*, a simple colotomy above the point of stenosis is indicated. Local anesthesia may even be employed.

Adhesions should be severed and tumors compressing the bowel removed. If the chronic obstruction be due to any of the causes which may also produce acute obstruction, appropriate surgical measures are indicated.

Early operation is preferable in most cases of chronic obstruction.

In acute obstruction engrafted on chronic stenosis there should be immediate resort to surgery.

# CHAPTER XXXIII

# NERVOUS DISEASES OF THE INTESTINES

UNDER this heading are included those conditions due to perversion of the innervation of the intestines independent of anatomic lesions of the intestinal wall or of distant organs.

In many cases the neurotic manifestations in the intestines are an independent manifestation of some general neurosis, such as of neurasthenia, hysteria, or hypochondriasis. Some few cases result purely from functional perversion of the intestinal nerves.

Intestinal neuroses may be divided into motor, sensory, and secretory. They often exist in combination. Psychic influences, such as fear, fright, worry, and anxiety, may be causes, as may reflexes from some diseased organs, such as the stomach or genito-urinary tract.

The nerve-centers influencing peristalsis, Meissner's and Auerbach's plexuses, have been described in the chapter on Physiology of Digestion, Part I, under Nervous Control of Peristalsis.

Secretion seems to be dependent to a great extent upon the ganglionic plexus. Moreau[1] ligated an intestinal coil and severed all the nerves passing to it. In a few hours it was filled with fluid, showing amylolytic qualities and containing albumin.

After ingestion of food into the stomach, secretion takes place in the lower part of the intestines before the arrival of the chyme. This was demonstrated by Quincke and Demant.

Vasomotor filaments exist in the intestines as stimulation of the splanchnic causes contraction, and its section causes dilatation of the intestinal blood-vessels. They are also concerned with absorption.

Sensory filaments exist in the intestines, since stimuli of greater intensity than normal, such as the ingestion of beans or cabbage, may give rise to sensations of pain or pressure. Kast and Meltzer[2] have demonstrated experimentally that the sensation of pain exists in the intestines, and that laparotomy, under cocain, causes anesthesia of the intestines through the cocain being carried by the blood.

## MOTOR NEUROSES OF THE INTESTINES

**Peristaltic Unrest (Tormina Intestinorum).**—This condition consists in marked rotary or rolling movements of the intestines, so that they frequently become visible. It is usually seen in patients with hysteria or hypochondriasis. Occasionally it is an independent affection. It is almost exclusively seen in the small intestine.

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[1] Centralbl. für die Med. Wissensch, 186, No. 14.

[2] Med. Rec., Dec. 29, 1906.

Peristaltic restlessness, which accompanies complete or incomplete occlusion of the intestines, is not included herein. There is not the peculiar stiffening of the intestinal coils, as is present with stenosis, and other symptoms of that condition are absent. Occasionally the condition occurs in persons presenting no other nervous symptoms, as after the ingestion of highly spiced or indigestible foods, after the excessive use of tobacco, mental excitement, or too much brain work.

Clinically, there are rolling, gurgling, squelching noises in the abdomen of varying intensity. They can often be heard at some distance and are a source of mortification to the patient. Pain, as a rule, is not present. The movements of the intestines may occasionally become visible and palpable. Eructations sometimes occur when peristaltic unrest of the stomach is associated.

Attacks occur at irregular intervals and may take place during menstruation.

*Diagnosis.*—Stenosis of the bowel must be excluded. The nervous type of peristaltic restlessness of the intestines is readily recognized. Prognosis is favorable.

*Treatment.*—This should be directed toward the tone of the nervous system. Heat externally applied and the drinking of hot water during the attack are of value. Spicy and indigestible food should be excluded. Priessnitz compresses should be applied to the abdomen at night. The bromids, valerian, and asafetida are useful. Arsenic alone or combined with iron should be employed in anemia. If there is any disturbance of the bowels (diarrhea or constipation) it should be properly regulated. Rarely a small dose of opiate, alone or combined with belladonna, is required. If the attacks occur at night, chloral hydrate, 15 grains (1.0), or veronal, 7½ grains (0.5), sulphonal or trional, 10 grains (0.6), may be necessary. Electricity and massage have been recommended. Change of climate is beneficial.

**Nervous Diarrhea.**—These exaggerated peristaltic movements occur not only in the small, but also in the large intestine. They may be limited to the colon in some cases. There is an increased transudation of fluid due to nervous influences. The reader should refer to a description of this condition under Diarrhea. Spastic constipation, due to local enterospasm and also spasm of the sphincter, sometimes occurs in neuropathic, hypochondriac, or hysteric subjects. These conditions are described under Constipation.

**Paralysis of the Intestines.**—Paralysis resulting from a mechanical obstacle to the passage of the intestinal contents has been described. Primary paralysis of the intestines without any organic obstacle will itself cause symptoms of obstruction. The reader is further referred to Dynamic Ileus.

There are several forms of this condition: 1. An intestinal coil may become paralyzed after forced reposition of a hernia or after incarceration; it may be due to direct traumatism, to abdominal operation, or to inflammation or ulcerative processes of the intestines.

2. It may result from reflex irritation of the inhibitory nerves of the muscular coats of the intestines, especially where there is injury or inflammation which does not necessarily involve the bowel.

Toxemia may be a factor. Contusion of the testicles, abdominal abscess, anesthesia, uremia, etc. are causes.

3. Neuroses, melancholia, hypochondria, or affections of the nervous system, such as meningitis, brain tumors, tabes, myelitis, etc., are also causes. Atony of the intestines, leading to coprostasis, has been suggested as a cause of intestinal paralysis. It would seem that the symptoms are produced by occlusion. Intestinal atony is really a subparetic condition, and the nervous type is described under Chronic Constipation.

Meteorism in hysteria is probably due to sudden paresis of the muscular coat of the bowel. See Meteorism.

*Treatment.*—Removal of fecal impaction by the fingers if present, enemata of soapsuds, 1 quart (litre), containing olive oil, 8 ounces (250 c.c.), and glycerin, 1 ounce (30.0), electric enteroclysis, enteroclysis, simple enemata, massage, and electricity are useful. With fecal impaction, liquid mercury, 10 to 20 ounces (300.0–600.0), given *through a stomach-tube*, is of value. It might otherwise enter the larynx. Various cathartics, such as castor oil, 1 to 2 ounces (30.0–60.0); olive oil, 4 ounces (128.0); physostigmin sulphate, $\frac{1}{100}$ to $\frac{1}{60}$ grain (0.0006–0.001), may be employed. Lavage, followed by the administration of the cathartic through the stomach-tube, is useful.

For further treatment, the methods pursued in Dynamic Ileus should be consulted.

**Paralysis of the Sphincters.**—It occurs as one of the symptoms of rectal affections. Tenesmus may lead to exhaustion. Ulceration and infiltration of the rectum at times involve the sphincters, interfering with their function or destroying it.

Improper methods of operations on the rectum may cause paralysis. Accumulation of feces may impair the tone of the muscles. Diseases of the brain and spinal cord may cause paralysis of the sphincters. *It may be a pure neurosis.*

Some patients are not able to keep the rectum tightly closed and a small amount of discharge continually escapes. In others involuntary movements occur after excitement, exertion, or during urination, there being only a partial paresis. With complete paralysis, flatus and feces escape involuntarily, even when resting.

With paralysis resulting from proctitis, hemorrhoids, stricture, etc., there is a continuous dripping of mucous secretion which irritates the skin.

*Diagnosis.*—The anus appears patulous and several fingers can be introduced into the rectum without resistance. To *diagnose purely nervous paralysis, anatomic lesions must be excluded by means of examination with a speculum.*

*Prognosis.*—This depends upon the cause—in the pure neuroses it is favorable.

*Treatment.*—Thorough evacuation of the bowels, preferably by enemata twice daily, is important. The addition of alum, 1 dram to 1 pint (4.0–500 c.c.) of water by enema, is useful. If due to nervous conditions, electricity and massage, especially local vibrations, as suggested by J. P. Tuttle, are beneficial. Tonics, such as iron and arsenic, are useful. Strychnin, $\frac{1}{60}$ to $\frac{1}{50}$ grain (0.001–0.0015) by hypodermic into the anal folds, has been recommended by Rosenheim. General improvement of the nervous system and at times change of scene are indicated.

Occasionally difficulty in urination and straining may cause paresis of the sphincter. Catheterization will improve this condition.

In the cases in which the nervous condition is *not responsible,* but *some anatomic lesion,* appropriate treatment is indicated.

## SENSORY NEUROSIS OF THE INTESTINES

I agree with Riegel that true colic is not a sensory neurosis. I have called attention to the fact that the pain of colic is produced by tetanic contractions of the intestinal muscles, and that it is a secondary symptom.

**Hyperesthesia of the Intestines.**—Under normal conditions digestion is carried on without producing any sensation whatever. In cases of neurasthenia, hysteria, and hypochondriasis the patient may be conscious of abnormal sensations in the intestines after the ingestion of food. They may occasionally appear after violent emotion or shock. These sensations consist in a feeling of fulness, stabbing, burning, tearing, and as if the ingesta were moving about in the abdomen. Occasionally delusions may develop. In some there is local hyperesthesia, especially in the rectum. There is a feeling of tenesmus or fulness, as if some foreign material were impacted therein, though the rectum is normal and contains no fecal matter. In others, pressure and weakness occur in this region, or there is burning, tickling, itching, stabbing, or a cutting feeling, at times combined with voluptuous sensations.

**Anesthesia of the Rectum.**—In these patients the desire for defecation is absent. In pronounced cases movement may occur without being felt. Such conditions are met with only in patients with spinal and brain trouble, or in the old and decrepit. Paralysis of the sphincters may occasionally accompany this condition.

*Treatment.*—This must be directed toward the improvement of the nervous condition. Change of climate and hydrotherapy are valuable. Highly spiced food, alcohol, and red meats should be forbidden.

Abnormal sensations in the rectum may be improved by cold rectal douches, sitz-baths, the cold prostatic cooler, such as I advocated in the treatment of hemorrhoids, and by rectal galvanization.

With rectal anesthesia cleansing enemata are useful. It may be necessary to wear a rectal obturator (Fig. 314) to prevent soiling.

**Nervous Enteralgia (Neuralgia Mesenterica).**—Aside from enteralgia due to irritating factors, it may result from a perverted state of the sensory intestinal nerves. This condition is not due to spasm of the intestinal muscles, like colic, but to a neuralgic affection of the bowels. It appears as a primary affection and is found in patients troubled with hysteria, neurasthenia, or spinal difficulty. It may occasionally be reflex, from abnormal conditions of the kidneys, bladder, uterus, ovaries, and liver. It may occur as a neuralgic condition even after the removal of some primary cause in the intestines.

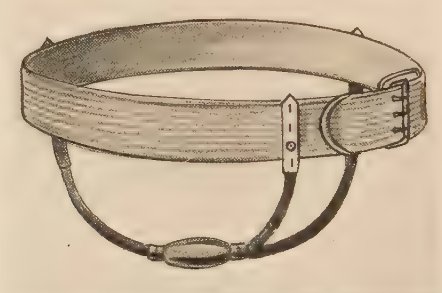

Fig. 314.—Rectal obturator.

The first symptom is pain, which usually begins in the umbilical region, mild at first, but gradually increasing in intensity. It may radiate in various directions. Pressure over the abdomen and the passage of flatus usually relieve the pain. It may be of a cutting or stabbing type, and may even produce shock or syncope. The bowels may be nearly normal or constipated. Appetite and digestion may be good. Palpitation, dyspnea, strangury, etc. may occur.

Neuralgic attacks can occur in lead-poisoning without the true spasm. Hemmeter has reported 3 cases of gouty neuralgia of the intestines. Romberg holds that the abnormal crisis of tabes dorsalis is due to nervous enteralgia. Examination of the symptoms demonstrates that true colic is present. The Romberg symptom, Argyll-Robertson pupil, and absence of patellar reflexes are diagnostic.

*Hypogastric Neuralgia* (Romberg).—In some cases of tabes there is a purely local form of neuralgia, limited to the rectum. The attacks are characterized by violent tenesmus, paroxysms of pain, a feeling as if a red-hot iron were inserted in the rectum, and occasionally diarrhea. This condition is also frequently found in diabetes and in women having uterine trouble, piles, or who are neurasthenic.

The picture presented by nervous enteralgia, on the other hand, as Riegel remarks, may occupy an intermediate position between peritonitis and colic (pseudoperitonitis). Violent attacks of pain occur in the abdomen at short intervals. Associated with this is frequently pronounced collapse, with great abdominal tenderness on light pressure. Vomiting, which is usually present in peritonitis, is always absent in these cases. The skin of the abdomen is generally hyperalgesic. The functions of the intestines and stomach between attacks are undisturbed, and the patients feel perfectly well.

*Treatment.*—This should be directed toward improvement of the hysteria and neurasthenia. Change of climate, hydrotherapy, massage, electricity, and moral treatment are of service. Arsenic is valuable. The bowels should be kept regular and a simple diet advised.

With neuralgia hypogastrica, if there is local disturbance, this should be treated. Warm sitz-baths and hot enemata are useful. Occasionally an opium-and-belladonna suppository may be required. Tabes should receive treatment.

## SECRETORY NEUROSES OF THE INTESTINES

Though secretion in the intestines immediately follows the entrance of food into the stomach, thus demonstrating the presence of secretory nerves in the intestines, we still have little knowledge of the subject.

Nervous diarrhea, which has been described as a motor neurosis, is often accompanied by an increased flow of intestinal juice. Increased intestinal secretion is found in membranous enteritis, though I do not consider this disease *a pure neurosis* of secretion.

## INTESTINAL NEURASTHENIA

Combinations of the intestinal neuroses frequently occur. Rosenheim designates such cases as intestinal neurasthenia. The appetite is good and the symptoms usually appear when intestinal digestion takes place—about two to three hours after meals.

There are pressure, tension, and griping in the abdomen. Occasionally there is nausea; and at times an evacuation of the bowels occurs accompanied with painful sensations in the abdomen and anus. Palpitation occurs at times; sometimes flashes of heat or cold. Generally the patient feels worse when resting in the recumbent position than when walking about. The symptoms usually disappear in a couple of hours to return later after a meal. Constipation usually is present.

The quality of the food *does not exert any influence on the symptoms.* Borborygmi and diarrhea occasionally are present, and the latter in the middle of the night or in the early morning. Indigestible foods are often well borne, while at other times small meals consisting of light food cause severe symptoms. Gastric neurasthenia is sometimes associated. This condition is found among the hysteric and neurasthenic.

**Diagnosis.**—Anatomic lesions causing these symptoms, intestinal dyspepsia, and enteroptosis must be excluded.

**Treatment.**—The general nervous condition must be toned up; iron, arsenic, strychnin, and the bromids are indicated, and ample feeding is required. Indigestible substances should be avoided, also red meats, to lessen the nervous irritability. The sour milks, such as bacillac, matzoon, kefir, etc., are of value.

## MUCOUS COLIC (MEMBRANOUS ENTERITIS)

Among the best-known symptoms for mucous colic are mucous colitis, membranous colitis, membranous or pseudomembranous enteritis, and tubular diarrhea. In all, there are about twenty-five names for this condition.

**History.**—Although no distinct accounts of this disease occur in the writings of the ancients, yet there may be detected some of its peculiar features in the description of certain pathologic conditions grouped under diarrhea, dysentery, etc. J. Mason Good, in 1825, was the first to classify the disease, calling it tubular diarrhea. Woodward described it in the " Medical and Surgical History of the War of the Rebellion." Siredy contributed a valuable paper in 1869. DaCosta, in 1871, described the nervous elements of the disease, stating that the condition is not a true inflammation. Leyden's work, in 1882, gave further stimulus to investigation, especially regarding the character of the dejecta. Nothnagel suggested the name " mucous colic," in order to show that a true enteritis need not exist. Mucous colic is, therefore, an entity, and may be defined as a " condition characterized by the excessive production of mucus in the colon, by attacks of painful spasms of varying degrees of severity and frequency, accompanied or followed by the expulsion of mucus in gelatinous masses, or in the form of tubular casts, or in tape-like pieces or strings, and, furthermore, characterized by anomalies of the gastro-intestinal functions and by various nervous symptoms."

**Age and Sex.**—It is a comparatively rare affection, occurring most frequently in women from twenty to forty, frequently in middle life. A few cases occur late in life and rarely in children. Boas reports one in early infancy. About 75 to 85 per cent. occur in women.

**Etiology.**—Space will allow me to mention only a few of the chief investigators. Among the various theories regarding the etiology, we may mention the following:

1. Neurasthenia is the prime factor—mucous colic is a secretory neurosis. Among the advocates of this view are notably DaCosta, Siredy, and W. Mendelson, of New York City.

2. The anatomic origin. Ewald lays stress on ptosis of the colon; Boas, on atony; Glénard, on splanchnoptosis.

3. Partly nervous and partly anatomic origin. Mathieu considers it a hypersecretion of mucus in patients of a neuro-arthritic type, who suffer from enteroptosis, intestinal sand being present. Hemmeter believes that often there is some connection with arthritis. Von Noorden lays stress on long-continued constipation in nervous subjects. Einhorn places it among the neuroses, but finds that it is associated in many cases with Glénard's disease (with gastroptosis and enteroptosis), and that achylia gastrica is present in many patients.

4. Tumors, adhesions, enlarged prostate, and various other factors are given. J. P. Tuttle believes mucous colic due to organic causes. Roger traces the cause to the liver, believing there is an anticoagulant in healthy bile, and when its production is interfered with by visceral ptosis, abnormal accumulation of mucus begins.

Nepper[1] also imputes the condition to disturbance of the biliary functions.

**Pathology.**—Necropsies are rare unless death results from some intercurrent disease. Autopsies in the cases of O. Rothmann, Osler (Edwards), and Weigert demonstrate that no inflammatory condition existed in the colon. There was simply hypersecretion of mucus. The consensus of opinion is that no inflammation exists. On the other hand, M. Rothmann reports 1 case and Hemmeter 2 cases in which, in addition, some catarrhal inflammation was present. Nothnagel explains this unquestionably by the fact that there are two classes of cases, one in which there is the pure "mucous colic," with hypersecretion of mucus; and the second class, in which the mucous colic is engrafted on a catarrhal colitis. I have noted, in my own experience, that the catarrhal colitis may be of such a mild type that attention may readily be diverted from it on account of the predominance of the symptoms of the mucous colic.

The mucus may be passed in the form of long, thin bands, ribbon-like or in the shape of a tapeworm; they may be tubular or form a cast of the intestines; in some cases these are of considerable length, several feet; the mucus may be in jelly-like masses or even in shreds, occasionally streaked with blood. This discharge should be carefully differentiated from fascia, tendons, the membranes of oranges, etc. After first treating with sublimated alcohol and then staining with Ehrlich's triacid solution, *a green color occurs with mucus*, of which this discharge consists; with fibrin it turns red. The color of the membranes in mucous colic is ordinarily grayish, though they may be translucent or even transparent. Microscopically, the membrane consists of a structureless matrix, with columnar epithelium scattered therein; its chief constituent is mucus.

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[1] Mucomembranous Colitis, its Causes and Mechanism, New York Med. Jour., May 23, 1908.

**Symptoms.**—These patients are markedly neurasthenic and morbidly self-conscious; in appearance they are usually emaciated, with a history generally of considerable loss of weight. There has been obstinate constipation of long duration, with an occasional intermittent diarrhea. Palpitation, dizziness, disturbances of the genito-urinary system, hysteric symptoms, anemia, headache, and gastric disturbances of various types are present.

On palpation of the abdomen sensitive points will often be detected. Patients give a history of a sudden attack of acute abdominal pain like severe colic, and the abdomen may become swollen and tense. At this time the nervous symptoms become extremely aggravated. Finally, the passage of the mucous masses described occurs spontaneously, with great straining or with artificial aid. These attacks occur with varying frequency and severity. Between the attacks the nervous condition of the patient may be slightly improved. This is the type of uncomplicated (pure) mucous colic.

Nothnagel describes a second type of enteritis membranacea which is engrafted on a colitis. He notes two classes of cases:

(*a*) That in which the severe *cramp-like attacks are absent;* the patient passes mucus continuously, with occasional tube-casts—a cystic colitis (Abercrombie's case), as shown at autopsy. This is not a true mucous colic.

(*b*) A class in which mucous colic is engrafted on a chronic catarrhal colitis—the latter due in this case to adhesions from recurrent appendicitis. There were small amounts of mucus passed at frequent intervals with occasional attacks of mucous colic. Operation relieved both conditions at first, but the mucous colic later returned.

I have had such a patient under treatment; the appendix had been removed and adhesions broken up. The patient improved for a time, but later relapsed. I found enteroptosis associated with gastroptosis. I applied Rose's belt and instituted treatment. Improvement immediately followed, with ultimate cure.

**Researches.**—For some years the author has carried on investigations in gastroptosis and enteroptosis at the Manhattan State Hospital, as well as other institutions, and also into the etiology of mucous colic,[1] and he is thoroughly convinced that enteroptosis is a factor in mucous colic.

In Fig. 315 is illustrated ptosis of the colon, narrowed at one point and sacculated above this. Gastroptosis is associated with it. This misplacement of the colon undoubtedly favors circulatory and, hence, secretory changes in the sacculated portion of the colon. Fecal accumulation is also favored, a further cause of irritation. Absorption, with resulting auto-intoxication and nervous disturbances following the same, can thus readily result. Naturally, a patient of nervous temperament, and there are undoubtedly many such, may be more markedly affected, but I do not believe that neurasthenia *per se* will cause that peculiar entity known as mucous colic any more than it will cause gonorrhea. There must be other factors.

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[1] Amer. Med., vol. ix, No. 9, pp. 349–354, March 4, 1905.

We know that mucous colic occurs in Glénard's disease, that Ewald believes that ptosis, and Boas that atony, of the colon are important factors, and Einhorn finds a large percentage of patients with mucous colic that have enteroptosis associated with gastroptosis and achylia gastrica.

I believe that mucous colic has as its chief etiologic factor ptosis of the colon with associated gastroptosis.

I have had under observation a patient with typic attacks of mucous colic which began one month after confinement. She had enteroptosis and gastroptosis (Landau's disease) due to insufficient support of the abdomen after the birth of her child. She was not neurasthenic, and was only nervous at the time of her attack.

A specimen of the mucus passed by this patient was about 12 inches long, flat, and tape-like, and gave the typic reaction to the mucin test; no fibrin was present. To my knowledge, this is the first case of mucous colic that has been *reported without the usual accompaniment of neurasthenia*,[1] and it substantiates my views. In addition, in 9 cases of mucous colic which I have carefully examined since I began these special investigations, I have found in every case varying degrees of gastroptosis with its associated enteroptosis.

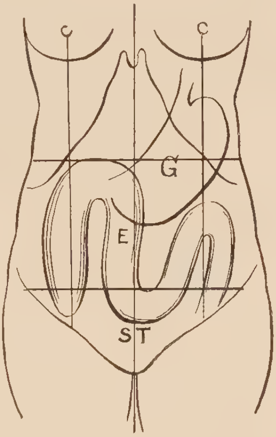

Fig. 315.—Mucous colic: Presence of gastroptosis (G) even of a mild degree, as demonstrable by gastrodiaphany shows enteroptosis (E) is present also; enteroptosis with sacculation and narrowing at ST; passive congestion and mucus accumulation occurs in enlarged (sacculated) portion of colon; mucous colic attacks occur as result.

In 4 cases there was hyperacidity; 2 cases, anacidity; 3 cases, achylia gastrica. Since the publication of the above article the author has investigated many more cases, all of which substantiated this view.

In the newborn and in young children, in whom several cases of mucous colic have been reported, neurasthenia surely cannot be claimed as a cause of the condition. As before stated, *visceral ptosis* may be present and the patient be in perfect health; but some contributory factor, local irritation, anemia, or intercurrent disease may destroy the equilibrium, and gastro-intestinal disturbances, constipation, etc. may result, and finally, mucous colic. On the other hand, gastro-intestinal ptosis may be brought about by loss of weight or other factors mentioned under Etiology of Gastroptosis, and mucous colic finally result. Enteroptosis is not invariably productive of mucous colic, any more than is typhoid always complicated by hemorrhage or perforation.

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[1] Med. News, Aug. 6, 1904; Amer. Med., March 4, 1905.

Enteroptosis with associated gastroptosis with gastro-intestinal disturbances I consider factors in the production of mucous colic, and the neurasthenia the result of auto-intoxication. In effect, it may be considered as one of the manifestations of Glénard's disease. Other contributory factors, such as rectal irritation, associated colitis, etc., will be referred to under Treatment. In a paper entitled "A Consideration of the Etiology of Mucous Colitis," by John A. Lichty,[1] there are reported 21 cases of mucous colitis; ptosis of the viscera was demonstrated in 16 cases; the other patients were seen before the author's attention had been directed to splanchnoptosis. He states that it is a well-known fact that not infrequently during the examination of a patient a condition of ptosis is found without any symptoms referring to it. In such cases there has been established what may be called a condition of perfect compensation, and physiologic function has not been disturbed. When, however, this compensation is lost or disturbed, the symptom-complex of mucous colitis appears. He notes a lithemic condition in several patients—notably one having had several attacks of acute articular rheumatism. The gastric secretion was studied in 8 patients—in 4 it was hyperacid; in 2, normal; in 1, hypo-acid, and in 1, achylic. G. H. Koehler[2] reports a case of mucous colic clearly dependent on gastroptosis.

**Prognosis.**—These cases require tact and patience, and most of them are of long duration. With perseverance, I believe them to be curable.

**Treatment.**—This may be summarized as follows: During the attack, rest in bed; the application of heat to the abdomen by flaxseed poultices, turpentine stupes, or hot pepper poultices—1 dram (4.0) red pepper to 1 pint (500 c.c.) of boiling water—a flannel being wrung out therein, covered with oiled silk, and applied to the abdomen. Dry heat, by means of a hot-water bag, salt-bag, or light tinplate (pie plate), heated in the oven and covered with flannel, may be employed. Spice poultices are of service. Moist heat, however, seems best.

The greatest relief to the cramps and bearing-down pains is afforded by enteroclysis by recurrent irrigation with normal saline solution at 110° to 120° F., oil of peppermint, 5 to 15 minims (0.296–0.888) to the quart (liter), may be added. Several gallons should be employed once or twice in twenty-four hours, and no fluid should be left in the bowel after irrigation, lest further cramps ensue. High enemata of warm olive oil—1 pint to 1 quart (500 c.c. to 1 liter)—are also of service, as they aid in relieving spasm just as does the internal administration of olive oil in spasm or stenosis of the pylorus.

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[1] Amer. Med., Aug. 6, 1902.

[2] Med. Sent., June, 1909, Portland, Oregon.

Hot saline rectal injections containing 2 to 4 ounces (60.0–125.0) of milk of asafetida may be employed.

*Diet.*—Fluid diet, milk, broths, soups, etc. should be enjoined during an acute attack.

*Medication.*—Tincture of belladonna in doses of 10 minims (0.592 c.c.) every three or four hours, and pushing even to physiologic symptoms, has given me the best results in the treatment of spasm. Occasionally it may be necessary to employ codein in 0.016 to 0.03 gram ($\frac{1}{4}$–$\frac{1}{2}$ gr.) doses, or even morphin, 0.008 to 0.016 gram ($\frac{1}{8}$–$\frac{1}{4}$ gr.), in conditions of extreme pain. If the acute attack is rather prolonged, the internal administration of valerianates or of asafetida, and the addition of milk of asafetida to the enema may prove to be of value.

Between attacks I apply proper abdominal support. For this purpose a silk elastic abdominal supporter, the Van Valzah-Hayes support, Gallant's or La Grecque corset, or Rose's adhesive plaster belt can be used. Rose's belt has the advantage of simplicity and it cannot slip or become displaced.

My great object is to "put on fat" in all cases, and as ptosis of the colon and of the stomach are great factors in the disease, the increase of intra-abdominal tension should be secured by this means. The belt is an aid in the relief of the functional disorders of the stomach incident to the gastroptosis. If the patient objects to the plaster, then the silk abdominal supporter may be employed. In exceptional cases it may be necessary to resort to the rest cure, associated with hydrotherapy and electrotherapeutics. Under such conditions we may increase the weight by following out Russell's method, such as he first instituted at the Post-Graduate Hospital in the treatment of tuberculosis. It was advocated by me in cases of gastroptosis. Regarding the constipation, the Küssmaul-Fleiner method of injecting into the rectum nightly or every other night warm olive oil, to be retained all night, is of great value. At the beginning one may employ a few ounces, increasing it to 1 pint (500 c.c.) or even 1 quart (liter). The patient should be taught regular habits in attempting bowel movement. A glass of hot or cold water administered an hour before breakfast is valuable as an adjunct. Fluidextract of cascara sagrada or the compound cascara tablets have been found serviceable, and in some cases sodium phosphate administered in the morning is of value. Other remedies, as suggested under Chronic Constipation, can be employed. A thorough bowel action should be secured daily. Massage of the bowel may be employed, massage with a cannon-ball, or vibratory massage. Sensitive areas must be avoided. These methods can be used while Rose's belt is *in situ*.

Enteroclysis several times a week with normal saline solution is useful, since it promotes intestinal peristalsis, prevents the accumulation of mucus, and lessens the chances of spasmodic attacks. With obstinate constipation electric saline enteroclysis is of value. In addition I sometimes employ baths, abdominal compresses, and electricity.

The carbonic acid bath (Nauheim) is of value in improving the circulation. For the nervous conditions the same bath (Triton salts) has proved of service. Achilles Rose has devised a simple method for administering the dry gas bath without the patient being obliged to disrobe (Fig. 316). Rose has recently improved on the dry bath apparatus, so it is possible to enter it on the floor level without climbing up steps.

The tank is filled with gas and the patient sits therein. The height to which the gas rises is estimated by means of a burning candle, which goes out when the gas reaches that point.

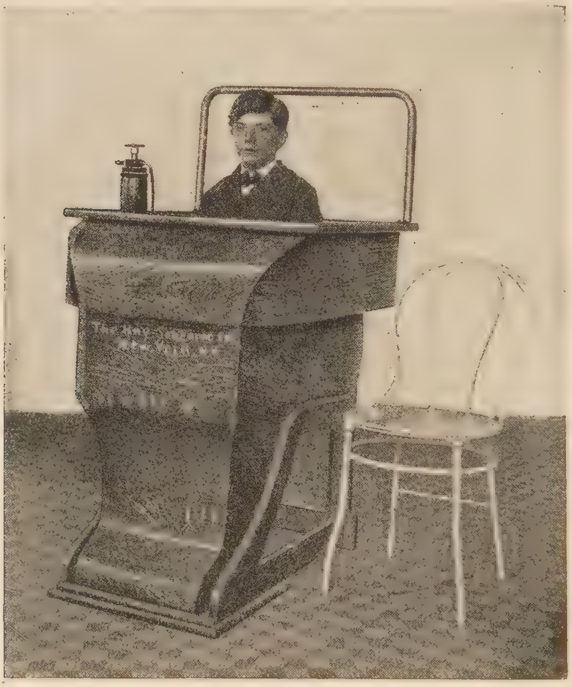

Fig. 316.—Rose's dry carbonic acid gas bath.

I have secured at least one brilliant result in the treatment of mucous colic by inflation of the colon with $CO_2$ gas, as advocated by Dr. Rose. The method seems in some cases to improve the local circulatory conditions in the colon, just as it affects the peripheral circulation when the bath is given. I believe it worthy of trial as an adjunct to the other treatment.

It merely requires a bottle with a large mouth. A glass tube passes through the cork. To this tube is attached a piece of rubber tubing with a rectal tip. From $\frac{1}{2}$ to 1 dram (2.0–4.0) each of bicarbonate of soda and tartaric acid are placed in the bottle, which is then filled two-thirds with water. The $CO_2$ gas is thus generated, and the bottle being elevated slightly above the rectum, the gas is allowed to flow in until slight distention is observed. This procedure can be carried out every other day. Rose's $CO_2$ bottle is illustrated in this volume.

I have referred to certain mixed cases in which there was a catarrhal colitis with a mucous colic later engrafted upon it. Among such we can classify those that may apparently be caused by excessive bicycling or horseback riding, enlarged prostate, uterine fibroids, adhesions from appendicitis, etc. In some of these conditions a local congestion of the rectum or sigmoid can be detected, and careful investigation will demonstrate that the attack first starts as a simple proctitis or colitis. Constipation has been previously present. Subsequent auto-intoxication, nervous symptoms, and mucous colic result. I believe that careful examination will reveal that these patients have had an existing ptosis of stomach and colon, quiescent, with no resulting symptoms, but as a result of irritation causing favorable conditions, mucous colic develops. The correction of such sources of irritation is undoubtedly rational, and will thus readily explain the improvement which at all times occurs after operative procedure, as in the chronic appendicitis case with colitis and mucous colic previously described. The existence of the "mixed cases" will undoubtedly "clear up" the hitherto apparently diverse opinions as regards the etiology of this disease.

In such cases, with a coexisting catarrhal colitis, irrigation with nitrate of silver,[1] 1.3 to 2 grams (20–30 grains) to 2 quarts of water, and followed by saline solution, or with resorcin, 0.65 to 1.3 gram (10–20 grains) in 2 quarts, or with listerin, borolyptol, glycothymolin, 4 to 8 grams (1–2 drams) to 2 quarts, or with gomenol, 4 grams (1 dram) in the same quantity of water, may prove to be of service. I have often found enteroclysis with demulcents, such as weak flaxseed tea, or 6 to 8 ounces (185–250 c.c.) of a saturated solution of gum-arabic added to 2 quarts (liters) of warm water, of value.

In pure mucous colic I employ only normal saline solution or the demulcents for removal of the mucus, since the condition is due to hypersecretion and not to inflammation. I should avoid silver irrigations in such cases, since I have already referred to the fact that irrigations of silver, tannin, alum, etc. can produce an artificial hypersecretion. Small doses of olive oil or of castor oil, in capsules, seem of value for constipation, and improve the tone of the mucous membrane of the intestines, providing they do not increase the patient's dyspeptic symptoms.

Extract of nux vomica, $\frac{1}{4}$ grain (0.016), or strychnin, $\frac{1}{60}$ grain (0.00108) t. i. d., is of service in increasing the tone of the gastro-intestinal tract and the general muscular system. Resorcin, 5 grains (0.3), or sodium benzoate, 5 to 10 grains (0.3–0.6), or bismuth salicylate, 5 to 10 grains (0.3–0.6), should be given if there is much gastro-intestinal fermentation. The use of the following, suggested by W. H. Thomson in the mixed cases for the treatment of catarrh, gives good results:

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[1] Protargol 1–2,000 is also useful.

| | | |
|---|---|---|
| ℞. | Silver nitrate | 0.32 gm. (gr. v); |
| | Resin of turpentine | 8.0 gm. (ʒij); |
| | Potash solution | 4.0 gm. (ʒj). |
| | Pulverized licorice, q. s. to make pills soft.—M. | |

Divide into 60 pills.

Sig.—Three pills t. i. d.

Copper sulphate, $\frac{1}{4}$ grain (0.016) t. i. d., may be substituted later; Fowler's solution of arsenic in 1-minim (0.06 c.c.) doses t. i. d. has also been found useful in these mixed cases.

**General Treatment.**—Exercise and outdoor life, as golf, etc., to strengthen the abdominal muscles, are important. During winter weather fencing is useful. The general nervous system must be toned up and anemia should be corrected. Iron tropon is easy to assimilate. An excellent combination is a fresh Blaud's pill (iron), 5 grains (0.32 grams), made soft with honey; in each pill is incorporated 2 minims (0.118) Fowler's solution of arsenic, and extract of nux vomica $\frac{1}{8}$ grain (0.008 gram), or sod. arsen., $\frac{1}{60}$ grain (0.00108), with $\frac{1}{60}$ grain (0.00108 gram) of strychnin. The glycerophosphates or phosphorus compounds are of value for the nervous conditions. Hydrotherapy, massage, and electrotherapy may be used.

*Diet.*—As before noted, fluid diet, milk, koumiss, bacillac, lactone-buttermilk, broths, gruels, etc., with the addition of somatose or liquid peptonoids, should be used during the attacks. Between attacks, Von Noorden advocates a very coarse diet (bread containing plenty of chaff, vegetables rich in cellulose, fruits with skins, etc.), to form ballast for the bowel. He claims excellent results. It is my *custom to determine the condition of the stomach.* Like Einhorn, I have found cases of achylia gastrica in mucous colic, but more cases of hyperchlorhydria and a few of hypochlorhydria. These special conditions should be treated in each individual case and appropriate diet instituted. Stomachics and dilute hydrochloric acid should be given when there is deficiency of HCl, and alkalis if there is hyperacidity (see Hyperchlorhydria, Achylia Gastrica, etc.). We should, however, give our patient abundant nutrition. Cod-liver oil and fats, such as Russell's emulsion, cream, etc., are of value when they can be assimilated. The addition of healthy fat, with increase in weight, means the cure of our patient.

*Surgery.*—Some writers, notably Hale White, have recommended a right inguinal colotomy to give rest to the colon in certain intractable cases. This would not relieve the ptosis, however. In severe cases one might resort to shortening the suspensory ligaments of the stomach and colon. Gastropexy and colopexy might be performed, but to my mind it is always objectionable to suture a viscus to the abdominal wall. If there is hepatoptosis, Elliot's operation for support of the liver might be instituted at the same time. These procedures will aid in the support of the floating kidney if such be present. Nephropexy, I believe, is rarely indicated when it is a part of general ptosis, unless there be some evidence of nephritis or interference with its functions. Some recommend a " revision " (tightening) of the abdominal muscles (recti) by means of suturing, so as to relieve the muscular relaxation. This last procedure, as advocated by R. T. Morris, I consider preferable. I believe that resort to surgery is rarely required except in the most obstinate cases, and only after at least two years' continuous medical treatment, with failure to secure cure or comparative comfort for the patient.

## CHAPTER XXXIV

# INTESTINAL PARASITES

Most of the animal parasites that occur in mankind inhabit the intestinal canal. There are about fifty varieties, but all do not produce morbid conditions. Some cause a pathologic state locally in the intestines or by their toxins in the blood. There are no absolutely characteristic symptoms produced by these parasites, but they are detected by discovering either them or, in the case of worms, their ova in the stools.

Gastro-intestinal disturbances, with or without anemia and with nervous symptoms, may result from their presence. There are two chief groups—the protozoa and the vermes. In passing, it is interesting to note that certain fungi have spread from the mouth to the lungs, in some cases causing symptoms resembling tuberculosis. Thus, Castellani[1] has reported tropical bronchomycosis with the oïdium albicans and saccharomyces fungi, found in the sputum in such cases.

## PROTOZOA

### AMEBÆ

In addition to the dysenteric amebæ which have been described, amebæ differing slightly in certain characteristics from the dysenteric variety have been reported. They are said to give rise to no symptoms or at times to slight diarrhea. Musgrave is skeptical as regards the existence of non-pathogenic amebæ.

### SPOROZOA

Coccidia are occasionally found in the stools. These are egg shaped, provided with a thin shell, are about 0.02 mm. long, containing in the interior a large number of nuclei, usually arranged in groups. They do not seem to have a pathologic bearing.

### INTERNAL PSOROSPERMIASIS

Psorosperms have been found in the liver, spleen, kidneys, and ileum, producing a disease similar to that in rabbits. One patient, notably referred to by Osler,[2] was thought to be suffering from typhoid fever. The patient had diarrhea and enlarged liver and spleen. Masses resembling tubercles in the liver, spleen, and ileum were found to contain coccidia.

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[1] Philippine Jour. of Sci., July, 1910.

[2] Prac. Med., 1906.

## INFUSORIA

**Cercomonas intestinalis** is pear shaped, has a distinct nucleus, and eight flagellæ. The head tapers obliquely and has a depression (Fig. 317). It is believed that this organism is liable to prolong existing catarrhal affection of the intestines.

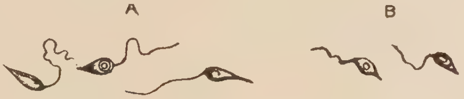

Fig. 317.—Cercomonas intestinalis: A, Larger, B, smaller, variety (Davaine).

**Trichomonas intestinalis** is distinguished from the former by its greater size and the row of fine cilia on its periphery (Fig. 318). In fresh dejecta its shows active movements. Zunker[1] reports it in mushy dejecta of yellowish-brown color and putrid odor. Dalley[2] reports a case of gangrene of the lung in which traumatism of the chest was a factor. Numerous trichomonas actively motile were found in the sputum.

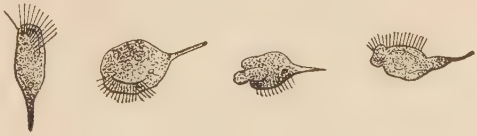

Fig. 318.—Trichomonas intestinalis (after Zunker).

**Balantidium (Paramœcium) Coli.**—The body is oval shaped, measuring from 0.07 to 0.1 mm. in length, by 0.05 to 0.07 mm. in breadth (Fig. 319). The anterior end is slightly truncated, with a short peristome, generally funnel shaped, and opens externally near the anterior pole. When feeding it opens out and broadens, so one can see it is a mouth which leads to a gullet and not a simple furrow. The left border has long cilia, while the rest of the mouth is destitute of them. The surface of the cortical layer is surrounded by a cuticle covered with cilia. The interior structure consists of granular substance. It contains a nucleus and contractile vacuoles.

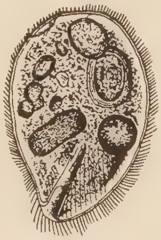

Fig. 319.—Balantidium coli (Malmsten).

Fat and starch granules and, occasionally, red and white corpuscles may be found within the granular substance. The posterior end is rounded and contains the anus. Particles may be observed to pass from it. The parasite can change its shape and possesses both forward and rotary motion. Reproduction occurs by division, budding, and conjugation.

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[1] Deuts. Zeits. f. Praktish. Medicin, 1878, No. 1.
[2] Jour. Amer. Med. Assoc., Oct. 15, 1910.

The balantidium is a parasite of the colon and cecum of *the hog*. Human infection probably occurs most frequently through the infusorium entering its host in the *encapsulated state*. When hog feces are dried and broken up, the encysted forms are scattered about and come in contact with the food or drinking-water. The parasite has been found in the city of London in the drinking-water. The disease frequently occurs after the preparation of sausage or the ingestion of uncooked sausage-meat. Malmsten first described the disease in 1857.

Musgrave has written on the subject, and for the most complete description the reader should refer to R. P. Strong's[1] article, "The Clinical and Pathologic Significance of Balantidium Coli." He tabulates 117 cases. Brewer[2] reports a case occurring in a child aged five years in the Philippines. Other intestinal parasites were also present.

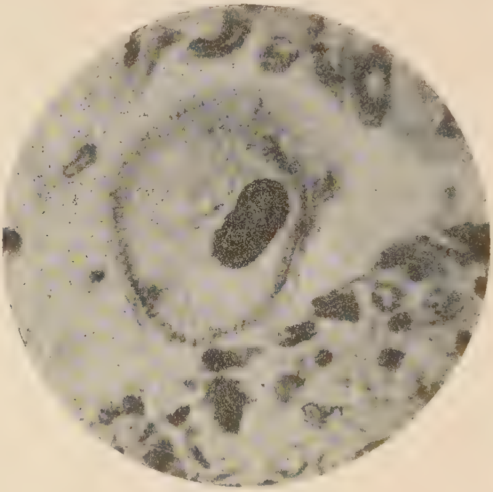

Fig. 320.—Balantidium coli: Parasite more highly magnified, showing flagella (Strong).

The balantidia frequently exist alone, though sometimes other parasites, such as Bothriocephalus latus, Ascaris lumbricoides, Trichocephalus dispar, etc., may be associated. Bowman[3] reports cases.

Harlow Brooks[4] found balantidia the cause of an epidemic of diarrhea among the orang-outangs in the New York Zoölogical Park. The lesions found in human beings seem to be an ulcerative colitis (Figs. 320–322), with the infection in the large intestine. In some cases there were swelling of the lymphatics of the mesentery and mesocolic glands and chronic adhesive peritonitis. Pulmonary, cardiac, renal, and cerebral complications may occur.

*Symptoms.*—The presence of Balantidia coli in the stools is usually associated with diarrhea; the feces are liquid, often contain mucus, sometimes undigested food, and frequently blood. Diarrhea is persistent until treatment is directed against the parasite. Colic is frequent, nausea and vomiting may occur. The abdomen may be swollen.

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[1] Bureau of Government Laboratories, Manila, Dec., 1904, No. 26.

[2] N. Y. Med. Jour., June 18, 1910.

[3] Jour. Amer. Med. Assoc., Dec. 2, 1911.

[4] N. Y. University Bull. of Med. Sci., Jan., 1902.

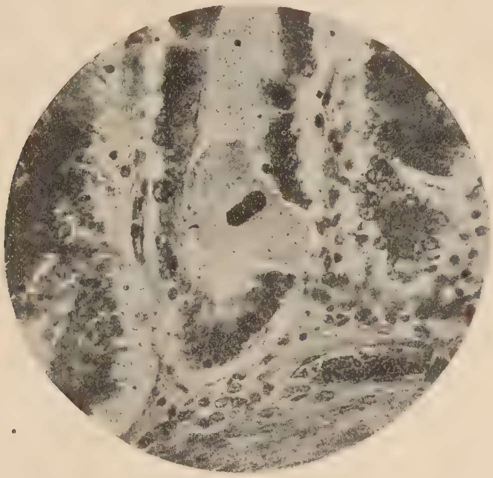

Fig. 321.—Balantidium coli: A parasite passing through the walls of a gland of Lieberkühn, rupturing the basement membrane. The parasite shows the striation referred to in the text (Strong).

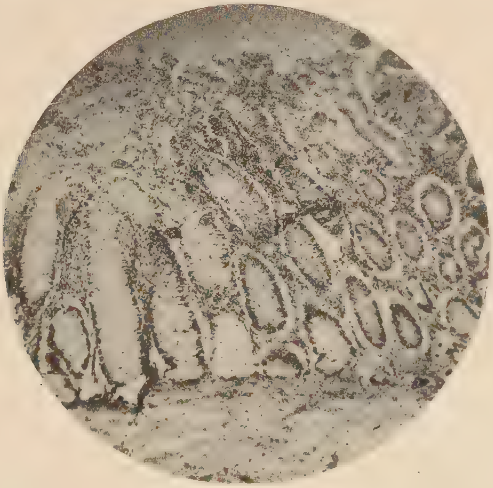

Fig. 322.—Balantidium coli. The early lesions of the mucosa, consisting of desquamation of epithelial cells of the glands, round-celled infiltration, etc. About a dozen parasites may be counted in this field (Strong).

It is often painful on pressure along the colon, and on palpation the latter may feel thickened. Tenesmus is common.

In chronic cases there are weakness, exhaustion, and emaciation; more or less anemia and edema of the feet and ankles.

In the fresh stools the balantidia move about rapidly, but die in from one-half hour to two hours after the dejecta have been passed.

Only 2 cases are reported in children; 25 per cent. of cases give the history of association with or caring for pigs or having eaten or prepared fresh sausage.

Eosinophilia is present locally in the intestines and also in the blood. The temperature may be subnormal or at times of considerable height.

The disease has been found in Germany, Sweden, Russia, France, the United States, and the Philippines. Balantidia probably occur more frequently than we suppose.

The mortality was 30 per cent., but depended somewhat on the presence of other diseases which were associated in many of the fatal cases reported. In 33 per cent. there were cures, and improvement in the other cases.

*Treatment.*—The following solutions for topical irrigations or enemata have been suggested:

*Ems' salt, by water enemata,* 15 grains (1.0) are added to 1500 c.c. (1½ quarts) of water, followed by quinin enemata, 1 quart (liter) of 1: 750 or 1: 500 strength.

Others suggest calomel, 2 grains (0.1) t. i. d. for two days only, or naphthalin, 5 grains (0.3) t. i. d., daily by mouth, with tannic acid, 1 dram to 1 pint (4.0–500 c.c.) of water, by injection, every day.

Acetic acid enemata—acetic acid, 50 grains (3.3) to 2 quarts (liters) of water at 37° C.—once or twice a day, with tannic acid 5 grains (0.3) t. i. d., by mouth, proved useful in some cases.

Another combination is salicylic acid, 15 grains (1.0), and sodium sulphate, ½ ounce (16.0), morning and night, by mouth, with enemata twice a day of salicylic acid (1: 1000), or salol, by mouth, 5 grains (0.3), four or five times a day, with salicylic acid (1: 1000), or boracic acid enemata 1 dram (4.0) to the quart (liter) of water, are suggested.

Sodium bicarbonate (2 per cent.) enemata, followed by a salicylic acid (1: 2000–1: 1000) enema, with salol, 5 grains (0.3), and tannalbin, 5 to 10 grains (0.3–0.6), by mouth, each several times a day, have given good results.

Thymol (1: 2500), acetozone (1: 1000), quinin bisulphate (1: 500), administered by enema or irrigation, as in dysentery, with the use of salol and tannin preparations by mouth, are valuable.

One might also administer small quantities of acetozone (1: 1000) by mouth.

## VERMES

**Cestodes (Tapeworms); Hydatid Disease.**—The adult parasites live in the small intestine of man; the larval forms, in the muscles and other organs.

The most important varieties of tapeworms found in human beings are the Tænia solium, Tænia mediocanellata, and the Bothriocephalus latus.

The symptoms produced by these varieties of tapeworms are about identical, except in the case of the bothriocephalus, which at times gives special symptoms.

The parasites are found at all ages, are not uncommon in children, and occasionally are found in sucklings. *They may cause no disturbance*, and one may only learn of their presence by noting segments of the tenia in the dejecta.

In other cases there may be general as well as intestinal disturbances.

There may be pressure in the pit of the stomach, abdominal pains, ravenous appetite (bulimia), nausea, at times loss of appetite, occasionally vomiting. Diarrhea is sometimes present or there may be constipation.

In women and nervous patients we may see mental depression and even hypochondria. There may be dizziness, headache, fainting spells, chorea, convulsions, and even epileptic attacks. Some patients may emaciate, feel weak, and suffer from palpitation.

The *bothriocephalus may cause a severe and even fatal anemia* (pernicious), with poikilocytosis and nucleated red blood-corpuscles; and with it there may be edema of the feet and eyelids and hemorrhages from the mucous membranes.

The metabolic products of this worm probably have a hemolytic action.

The *diagnosis can only be made by discovery* of the segments of the parasites or their eggs in the stools.

*General Description of the Tapeworm.*—It has a scolex or head, which may live for years even when detached from the rest of the body, an oblong neck, and detachable segments (proglottides). These vary in size and shape and possess the power of motion. The worm is flat and devoid of mouth or intestines. It grows by alternate generation through germination of a pear-shaped primary host (head), and remains united to the latter for a time as a colony of band-like shape. Each segment forms a sexually active individual. The proglottides gradually increase in size as they become more distant from the head, and then diminish again toward the extremity. The tapeworm is an hermaphrodite. On its head are four sucking disks, by which it attaches itself to the mucosa of the intestines. By means of pores it derives its nourishment from the chyme.

The older proglottides contain many fructified eggs. These are emptied at intervals into the intestinal canal and appear in the dejecta. The ovum contains an embryo, which requires for its development an intermediary host. After reaching the stomach the envelope is dissolved by the gastric juice. The embryo is set free and finds its way by the lymphatics or blood-vessels to some place (usually the muscles) where it settles. It here surrounds itself with a sac, which may later be surrounded by a calcareous deposit. In this condition it is called a cysticercus or measle. When the measle reaches the stomach of a new host it opens, and its scolex enters into the small intestine, where it develops into a full-grown tapeworm.

*Tænia Solium.*—Armed tapeworm or pork tapeworm. This is not common to North America, but more frequent in Europe and Asia. When mature it is from 6 to 12 feet (2–4 meters) or more long. The head is smaller than the head of a pin, spheric, and provided with four sucking disks, in the middle of which is the rostellum and a double row of hooklets, from twenty-four to twenty-six in number, and hence is called the armed tapeworm (Fig. 323).

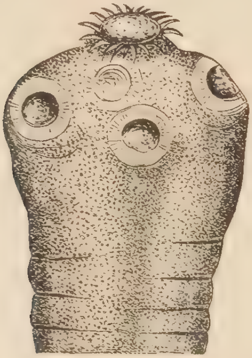

Fig. 323.—Head of Tænia solium, rostellum with hooks; suckers (Mosler and Peiper).

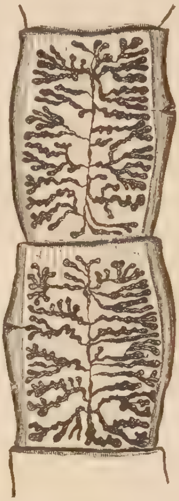

Fig. 324.—Mature segments of Tænia solium; proglottides; uterus and branches (Mosler and Peiper).

The neck is narrow and thread-like, nearly 1 inch long. The body is divided into segments, which possess both male and female generative organs, and at about the four hundred and fiftieth they become mature and contain ripe ova. The segments are about 1 cm. in length and from 7 to 8 mm. wide. The worm attains its full growth in about three to three and a half months, about which time the segments are continuously shed and appear in the stool. The uterus forms a straight median tube in each segment, giving off five to seven branches on each side. The branches are undivided at first, but finally ramify as a tuft (Fig. 324).

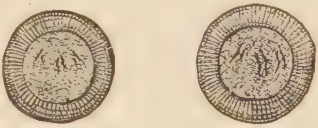

Fig. 325.—Eggs of Tænia solium, showing thick shell (Mosler and Peiper).

The eggs are rounded and provided with a thick shell (Fig. 325).

Rarely the cysticerci (measles) are found in man, as in the muscles, brain, eye, and skin.

In the muscular system they cause pain, numbness, weakness, and symptoms a little like peripheral neuritis. In the ventricles of the brain irritative symptoms may result. In one case diabetic symptoms were reported. They can be recognized in the eye. Tænia solium was formerly believed to exist alone, but several have been found together.

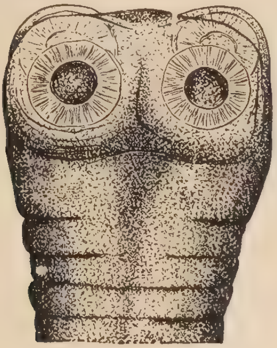

Fig. 326.—Scolex of Tænia saginata (Mosler and Peiper)

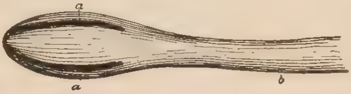

Fig. 328.—Bothriocephalus latus: *a, a,* Head; *b,* neck (Blanchard).

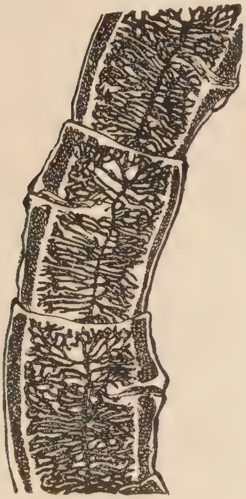

Fig. 327.—Segments of Tænia saginata (Mosler and Peiper).

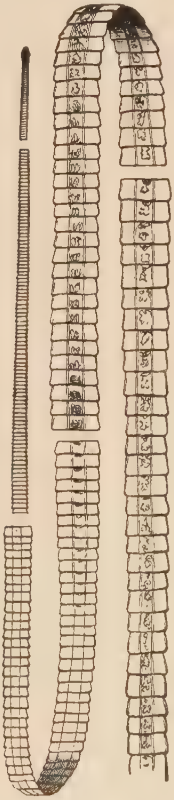

Fig. 329.—Bothriocephalus latus (Eichhorst).

*Tænia saginata,* or mediocanellata, the unarmed or beef tapeworm.

This is the most common form seen in America as well as abroad. It is longer, thicker, and wider than the Tænia solium. It may attain a length of 15 to 20 feet (approximately $4\frac{1}{2}$ to 6 meters) or more. The head measures over 2 mm. in breadth, has four large sucking disks, but no hooklets and no rostellum (Fig. 326). It is square shaped.

The ripe segments are from 17 to 18 mm. in length and from 8 to 10 mm. in breadth. The uterus consists of a median stem, with from about twenty to thirty-five lateral branches (Fig. 327).

The ova are larger and the shell thicker, and possibly slightly more elliptic, but the two forms are difficult to distinguish by their ova. The measles (cysticerci) occur in beef and are smaller than those of the Tænia solium. Human beings acquire this worm by the consumption of raw beef.

*Bothriocephalus Latus* (Tænia Lata, or Pig Head).—This is found in certain districts bordering on the Baltic Sea, in Holland, Switzerland, and Japan. A few cases have occurred in the United States, believed generally to have been imported.

The parasite is large and long, measuring 25 to 30 feet or, more approximately, $7\frac{1}{2}$ to 9 meters.

The head is elongated, almond shaped, being about 2 mm. long and 1 mm. broad; it has two grooves, probably suckers, on its flat surface (Fig. 328).

It has no hooklets. The neck is narrow and short, about 2 cm. in length, and passes at once into the body segment. The body is thin and flat (Fig. 329). The full-grown proglottides are nearly square and show the sexual organs in the center.

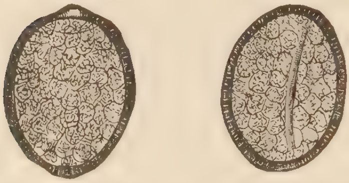

Fig. 330.—Eggs of Bothriocephalus latus (Mosler and Peiper).

The uterus presents as a median dark line, with four to six lateral branches, looking like a star or rosette. The eggs are oval, round, with a thin membrane and a lid (Fig. 330). They measure 0.07 mm. long and 0.04 mm. in width. The larvæ develop in the peritoneum and muscles of pike especially, and of fish such as the turbot, perch, and trout. Infection occurs through eating insufficiently cooked fish.

Tænia lata occur in the small intestine of men and rarely in dogs. A *severe and even fatal form of anemia* may result from this worm.

There are a few rare forms of tapeworms occasionally found in human beings.

*Tænia nana* (Hymenolepsis nana) occurs chiefly in Italy, occasionally in Egypt. It is the smallest tapeworm found in man, measuring 10 to 15 mm. long, and may have nearly two hundred segments. The head has four sucking disks, a rostellum, and about twenty-four hooklets in a single row. Proglottides are short and broad. It is found more frequently in children, and occurs in large numbers in the small intestine.

Nervous disturbances, fainting spells, and even epileptiform attacks are produced thereby.

The *Davainea Madagascariensis* (Tænia Madagascariensis) is a rare form of this worm.

*Tænia cucumerina* (Elliptica, or Dipylidium Caninum).—This is small, of cucumber shape, occurs frequently in the intestines of the dog, and has been found in small children. The larvæ develop in the lice and fleas of the dog. The worm is 10 to 40 cm. long and about 3 mm. wide.

*Tænia Flavopunctata* (Hymenolepsis Diminuta).—This has been met with in about 12 cases. The worm is 2 to 6 cm. long and about 3 mm. wide. Its head is small, club shaped, and provided with sucking disks. It is common in rats. The larvæ develop in Lepidoptera and in beetles.

*Bothriocephalus Cordatus.*—This tapeworm resembles the Bothriocephalus latus, except that it is shorter and the head merges into the proglottides without an intervening neck. It occurs in the intestines of men and dogs in Greenland.

Other types of tapeworms occur, but they are excessively rare and not found in Europe or America.

*Echinococci* are the larvæ of the Tænia echinococcus of the dog. The latter is a tiny cestode 4 or 5 mm. long, consisting of three or four segments, of which the terminal one alone is mature. The head is small, provided with four sucking disks, and a rostellum with a double row of hooklets.

As a result of the ingestion of these parasites, cysts develop in various parts of the human organism, as in the liver, muscles, etc. These cysts contain scolices, the head of the tænia presenting four sucking disks and a circle of hooklets.

Cysts have been passed per rectum. The disease is common to Iceland, not uncommon in Europe, but rare in this country. The reader is referred to Echinococcus Disease in any work on Practice of Medicine.

**Treatment of Tapeworms.**—To escape infection avoid raw or medium-done meats, pork, and fish. One should not trust to meat inspection alone. Thorough cooking of the meat is the only guarantee of extinction of the cysticerci. All worms or fragments removed should be destroyed by burning, and if one handles the proglottides or ova, the hands should immediately be disinfected; also the stools.

For about two days before administering a vermifuge the patient should be kept on a scanty diet, consisting of broths, soups or milk, with a few crackers. The night before, no food; and in the morning a cup of tea or coffee, followed in about an hour by the vermifuge.

Calomel, 5 grains (0.3), castor oil, $\frac{1}{2}$ to 1 ounce (15.0–30.0), or a saline cathartic should be given daily for a couple of days previously.

*Male fern is considered quite efficient.*

Extract of filix mas, ethereal, $1\frac{1}{2}$ to $2\frac{1}{2}$ drams (6.0–10.0), mixed with simple syrup; follow in a couple of hours by a saline cathartic or castor oil.

The following have also been suggested: Oleoresin aspidii (male fern), $\frac{1}{2}$ to 1 dram (2.0–4.0), in capsules, coated with keratin; a few hours later castor oil, 1 ounce (30.0).

*Filicic acid* (filmaron), an amorphous principle from root of male fern, insoluble in water. It should not be administered in fatty oils or alcohol, as they dissolve it and it is toxic. Give in capsules, $7\frac{1}{2}$ to 15 grains (0.5–1.0); follow by a saline purgative (not castor oil).

*Pomegranate root* is efficient, given as an infusion of the *bark;* 3 ounces are macerated in 10 ounces of water and then reduced to one-half by evaporation. The entire quantity is taken in divided doses within an hour or more. It is effective, though sometimes producing colic.

The active principle of the root, *pelletierin,* 4 to $7\frac{1}{2}$ grains (0.25–0.5), in sweetened water, to which tannin, 5 grains (0.3), can be added may be given as a substitute. These remedies are followed in a couple of hours by a purge. Pelletierin tannate can be secured and given in the same dose.

*Pumpkin seeds* (Semina cucurbitæ), 3 or 4 ounces (90.0–125.0), should be bruised and macerated for twelve to fourteen hours; then mixed with a little grape-sugar, diluted with milk, 1 pint (500 c.c.), and take in two doses about half an hour apart; then follow in two hours by castor oil.

*Turpentine,* Oleum terebinthinæ (spirits of turpentine), 1 ounce (30.0), in honey or with sugar, follow with a glass or two of milk; and two hours later a cathartic.

I have found *pine-needle oil* (Gardner's), 1 to 2 drams (4.0–8.0), also efficacious.

*Cusso* (kousso).—Cusso pulv., $\frac{1}{2}$ ounce (16.0) with mel depuratum (honey), $\frac{1}{2}$ ounce (16.0), is useful.

Or cusso can be mixed with sugar, water, or lemonade, and taken in divided doses within an hour. Though cusso is cathartic, it is preferable to follow in two hours with a dose of castor oil.

*Kamala.*—Pulv. kamala, 1 to 2 drams (4.0–8.0), suspended in syrup or in wine. This preparation is purgative and may cause griping, nausea, and vomiting. The dose should be distributed over an hour. Castor oil may be given later.

*Cocoanut* has been recommended as a vermifuge; the milk and albumin of an entire nut should be taken within an hour.

*Naphthalin,* in capsules, 10 to 30 grains (0.6–2.0), in divided doses, within a few hours, is another vermifuge.

*Salol*, 45 grains (3.0), in capsules, in divided doses, has been recommended.

A combination of these remedies is often effective. Thus: take ½ ounce (16.0) of an infusion of pomegranate seeds; pumpkin seeds, 1 ounce (30.0); pulverized ergot, 1 dram (4.0), and boiling water 10 ounces (300 c.c. approximately). Make an emulsion of male fern—1 dram (4.0) ethereal extract with acacia powder. Mix the emulsion and infusion and take fasting at 9 A. M. Follow a couple of hours later with castor oil or a saline cathartic.

Osler recommends the addition of croton oil, 2 minims (0.118), to the above, but I think this rather too active treatment. Male fern, pumpkin seed, pomegranate, and turpentine are the best remedies.

About two hours after the vermifuge a cathartic, such as citrate of magnesia, 1 to 2 drams (4.0–8.0), or magnesium sulphate, larger doses, or some other saline cathartic, or castor oil, 1 to 2 ounces (30.0–60.0), should be given.

The head of the tapeworm should be looked for, as the parasite will regrow if this is not removed. In some cases this is difficult to find. Children require proportionately smaller doses, according to their age. Care must be exercised with patients who are debilitated, recently convalescent from typhoid, or have severe intestinal disorders. It may be necessary to postpone treatment.

**Trematodes (Fluke Worms); Distomiasis.**—Flukes are *found in the lungs, liver, small intestine, and in the blood;* in the latter case affecting chiefly the urinary system and the rectum.

The trematodes are solid worms of leaf or tongue shape. They possess a clinging apparatus in the form of oral and ventral sucking cups, which vary in number. Sometimes they also have hook or clasp-like projections. The intestinal canal is without an anus and is split like a fork. They are generally hermaphroditic.

Flukes are found in China, Japan, India, Egypt, Arabia, and Persia, and imported cases have been found in Canada and the United States.

They have been found in the cat, dog, and hog in this country.

Five species of *liver fluke* are known to occur in man. The Distoma conjunctum, the Indian liver fluke, usually described, the Distoma lanceatum (lancet fluke), and the Distoma sinensis are the most frequent, occurring in the liver, the last being most important.

The Fasciola hepatica, common to ruminants, and the Opisthorchis (Distoma) felineus, occurring in Prussia and Siberia, and found in cats in Nebraska, are the other varieties.

In general, we may say the liver fluke is of leaf shape. It may vary in length from 10 to 20 mm. by 2 to 5 to 10 mm. broad. The cephalic end projects like a beak and has a small cup-like sucker, in which lies the mouth. Behind this, on the ventral surface, is a second cup, and between the two is a special orifice. The uterus appears as a convoluted bag behind the posterior sucker. On each side of the body are the ovisacs and the branched testicular canals (Fig. 331).

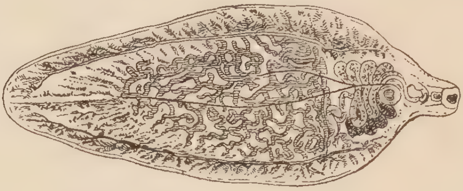

Fig. 331.—Distoma hepaticum, with male and female sexual apparatus; × $2\frac{1}{2}$ (Leuckart).

The eggs are oval, 25 to 30 $\mu$ long by 15 to 17 $\mu$ broad, of brown color, with a sharply defined operculum (lid) (Fig. 332).

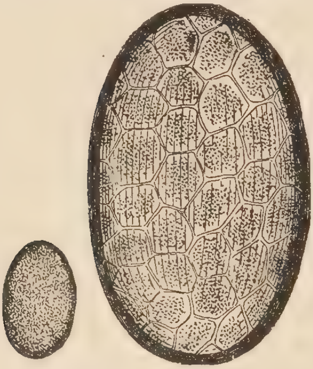

Fig. 332.—Eggs of Distoma hepaticum and Distoma lanceolatum, moderately magnified (Heller).

The *Distoma lanceolatum*, another variety of liver fluke, has a lancet shape and the head is not especially marked off from the body (Fig. 333). The eggs are rather small, 0.04 mm. long (Fig. 334).

Young children suffer more frequently from liver fluke; sometimes whole families or villages are attacked.

There is an irregular diarrhea; there *may or may not be blood.* The liver enlarges. There are often pain and an intermittent jaundice, but not much fever. Anasarca and ascites come on later. The ova of the parasite are found in the stool.

*Intestinal Distomiasis.*—In India the Distoma fasciolopsis has been found in a number of cases in the small intestine, with diarrheal symptoms.

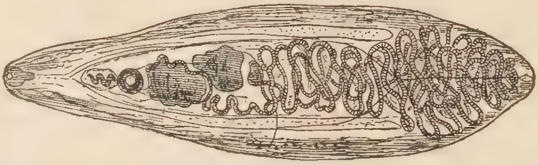

Fig. 333.—Distoma lanceolatum with its inner organs; × 10 (Leuckart).

*Hemic Distomiasis; Distoma Hæmatobium; Bilharziasis; Schistosoma Hæmatobium; Bilharzia Hæmatobia.*—Endemic hematuria, particularly in Egypt, had been known for many years, when in 1851 Bilharz discovered the parasite of the disease. The blood fluke prevails in South Africa (the Transvaal); in North Africa, *especially in*

*Egypt;* in Arabia, Persia, and the west coast of India. It is prevalent in Japan. It has been observed in Porto Rico and in the Philippines. It has been reported 104 times on the Isthmus of Panama according to statistical records from the Chief Sanitary Office from 1904–1909 inclusive. It has also occurred in Antigua, Trinidad, Demarara, Barbadoes, Jamaica, and Dutch Guiana.

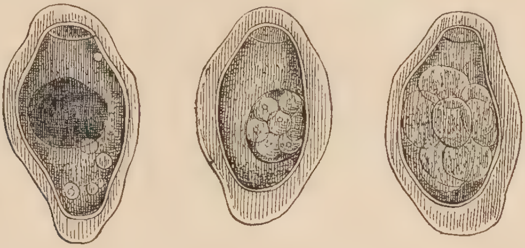

Fig. 334.—Egg of Distoma lanceolatum shortly after the formation of a shell; × 400 (Leuckart).

Brayton[1] reports several interesting cases from the Ancon Hospital on the Isthmus. The disease is rare in the United States, only 7 cases having been reported. In addition, 7 cases occurred among the Boers who were on exhibition in this country after the African War.

The first case of rectal infection reported in the United States was in a German, who evidently contracted the disease in Brazil and who was treated at the German Hospital. He suffered from mixed infection—Strongyloides intestinalis, Trichocephalus, and Schistosoma hæmatobium, reported by L. Blumgart,[2] of New York.

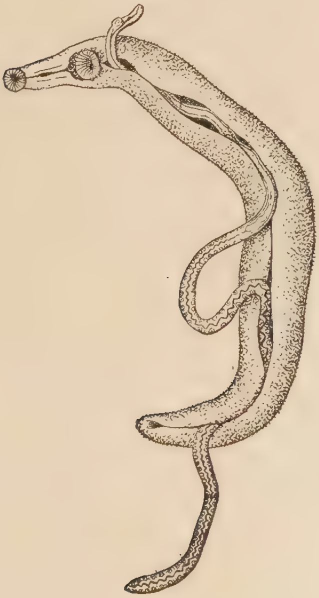

Fig. 335.—Male and female of Bilharzia hæmatobia (Loos).

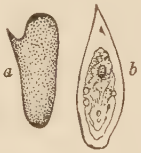

Fig. 336.—Eggs of Distomum hæmatobium (Bilharzia hæmatobia), length, 0.12 mm.; breadth, 0.05 mm.: *a*, Egg with lateral spine; *b*, egg with terminal spine; × 150 (after Bilharz).

[1] Jour. Amer. Med. Assoc., April 30, 1910.

[2] Med. Rec., April 6, 1907.

The Schistosoma has separate sexes and carries the female in a gynecophorous canal. The male is from 12 to 14 mm. long. Its body has ciliated warts on the integument, but otherwise is smooth, and in the posterior portion is rolled up into a tube, which serves for the reception of the female (Fig. 335).

There is a sucker at the anterior end and a second one posterior to it on the ventral surface. The female is from 16 to 18 mm. long and nearly cylindric. The eggs have a terminal or lateral spine (Fig. 336). The trematode is most abundant in the blood of the portal system, while the ova lodge in the capillaries, especially of the bladder, urinary organs, rectum, and lungs. In the stools of the case reported the spine was placed laterally on the ova.

Infection is now considered to take place in two ways—either by the gastro-intestinal tract, through infected food or water, or through the skin by bathing in infected streams.

The parasite reaches the portal system, where it develops. The males, bearing the females, creep to various parts, particularly the bladder and rectum. The eggs are laid in the tissues, but wander like other sharp foreign bodies, and escape with the urine and feces. The eggs in the tissues cause irritation, fibroid changes, and papillomata in the bladder and rectum. Hematuria and bladder irritation, chronic cystitis, tenesmus, mucus and blood in the stools, ulcerative proctitis, calculi in the kidney and bladder, peri-urethral abscess and perineal fistula, vaginitis, inflammation of the ureters and seminal vesicles may all occur. Bilharzial colitis has been reported. In the lungs the blocking of blood-vessels from the ova and the surrounding infiltration give rise to deposits which may be mistaken for tuberculosis. Cirrhosis of the liver and biliary calculi may be caused by the presence of the parasite.

Anemia is present and eosinophilia is quite marked.

In 1904 there was described a new blood fluke, Schistosoma Cattoi, or Japanicum, found in Japan. It lives chiefly in the vessels of the alimentary canal and ulcerative lesions are found therein. The ova appear in the feces. Katsurada has studied numerous cases. This condition is known in Japan as the "Katayama disease," from the name of a town in which it is quite prevalent.

Catto described certain bodies he found in a Chinaman in 1904, and the new fluke is sometimes called by his name.

Paul G. Woolley[1] has reported a case occurring in the Philippines, and has given an excellent description of the disease.

The worms are characterized by the *absence of ciliated warts on the integument,* which *are a feature of the Schistosoma hæmatobium.* The worm averages 10.43 mm. long. The eggs are smaller, brown in color, have blunter ends, and *no spine.*

Katsurada gives the most definite reports of the disease "Katayama," according to Woolley:

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[1] Philippine Jour. of Sci., Jan., 1906, vol. i, No. 1.

"Defective physical developments is the rule in the affected children. Diarrhea is usually the first symptom to be noted, while anemia and ascites generally follow later; the most striking feature is the shape assumed by the trunk. The hypogastric region seems to shrink, while the epigastric enlarges, a transverse furrow forming directly above the umbilicus, so that the general appearance of the abdominal region is that of an inverted gourd. Dilatation of the epigastric region and of the lower part of the thorax were noted even in patients whose liver and spleen were not much enlarged. The commonest symptoms are an initial increase in the size of the liver, followed by a decrease, a secondary enlargement of the spleen, a mucosanguineous diarrhea, severe attacks of ascites, and progressive anemia." Katsurada found the ova of the parasite under discussion, and also those of Trichocephalus dispar, Uncinaria, and Ascaris lumbricoides in the stools of his patients.

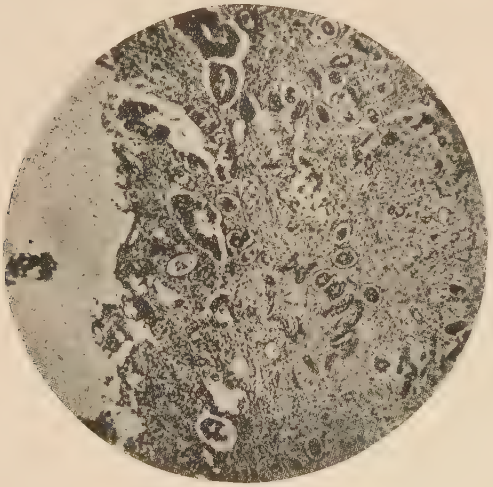

Fig. 337.—Schistosoma Japanicum. Ova in mucosa and submucosa of large intestine. Shows atrophic and infiltrated condition of mucosa. Hematoxylin (Woolley).

The rectum and appendix were the parts chiefly affected, but the ova were found in the subperitoneal layer, the submucosa, and mucosa, especially in necrotic areas, from the cecum to the anus. Adult trematodes were found in the blood-vessels.

In Woolley's case there were also amebæ and the ova of the uncinaria; but in the fibroid tissue of the submucosa of the large intestine there were many ova of the Schistosoma Japanicum surrounded by round-cell infiltration (Fig. 337). A type of cirrhosis was produced in the liver. Splenomegaly, ascites, dysentery (specific), and possibly Jacksonian epilepsy may be produced by these trematodes, according to Woolley.

The disease is probably water-borne, originating in rice-fields or irrigated gardens, from human fertilizer. Infection may occur through the skin or by the gastro-intestinal canal.

*Treatment.*—The extract of the male fern is recommended for distomiasis and the treatment as of tapeworm. Nothing has been found for the treatment of the parasite in the blood. The author would suggest the trial of urotropin, 10 grains (10.6), and sodium benzoate, 10 grains (0.6), three or four times or more a day. The latter lessens the irritation of the urotropin, as demonstrated by William H. Thomson.

The inflammation of the bladder, colon, and rectum should be treated as indicated by irrigation, etc. Solutions, as described under Proctitis and Colitis, can be employed for the latter complications.

**Nematodes (Round Worms).**—Round worms have a slender, cylindric, at times a filiform body, with neither segments nor appendages. The integument is thick and elastic. The mouth is at one extremity and furnished with either soft or horn-like lips. The alimentary canal extends through the entire body and terminates in an opening on the ventral side near the posterior extremity. The sexual organs and their orifices are on the ventral surface. The female aperture is at the middle of the body, while in the male the sexual orifice is near the anus. The males are usually smaller than the females.

**Ascaris Lumbricoides (Round or Spool Worm).**—This is one of the common parasites observed in man. It is cylindric in shape, pointed at both ends, and of a yellowish-brown or slightly reddish color. It varies from 4 to 12 inches (10–30 cm.) in length, the female being as large as 12 inches (30 cm.), while the male is only one-half or two-thirds the length, 8 inches (20 cm.), and frequently much smaller.

The posterior extremity of the male is bent in the shape of a hook and furnished with two spicules or chitinous processes (Fig. 338). The mouth has three muscular lips provided with very fine teeth.

The worm is transversely striated and has four *longitudinal bands*.

The sexual opening of the female is anterior to the middle of the body. The eggs when ripe have a double shell, and around this is an albuminous envelope, irregular in shape, studded with excrescences (Fig. 339). The long diameter of the ovum is about 0.075 mm. and 0.058 mm. in width. Atypic (unfertilized) eggs have been described by O. T. Logan[1] of China. When taken from the uterus it has not the typic thick shell, but is granular, elliptic, and enclosed in a delicate membrane (Fig. 340).

In the feces, the yelk is not finely granular, but coarsely globular; the albuminous coat is less voluminous and projects from the shell like blunt saw teeth. The unfertilized egg[2] is longer and narrower than the fertilized egg and markedly elliptic, with a tendency to flatten at one or both ends. It is occasionally oval, but never round.

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[1] N. Y. Med. Jour., Dec. 21, 1907.

[2] Ibid., Aug. 19, 1905 (Wellman); Reference Handbook Med. Sci., p. 502.

An irritating, odoriferous substance is formed by the round worm. Huber[1] states that it may occasion urticaria in those predisposed to this symptom. Peiper suggests that the nervous symptoms, sometimes resembling meningitis, may be due to this poison; and Chauffard and Marie report fever, intestinal symptoms, diarrhea of intermittent character and foul breath, so-called typholumbricosis, in connection with these worms. The fever may continue for a month or more.

The parasitic life history is direct, by ingestion of the ova, with no intermediate host. The parasite occupies the upper part of the small intestine. Usually not more than one or two are present, but they may occur in enormous numbers.

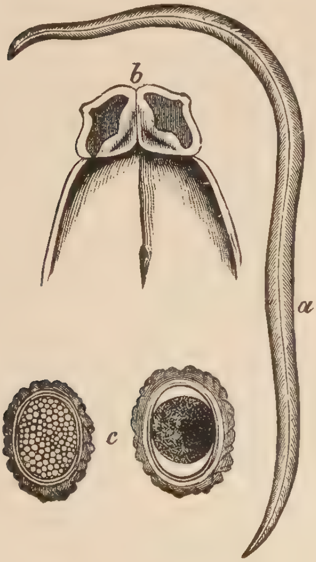

Fig. 338.—Ascaris lumbricoides: *a*, Body; *b*, head; *c*, eggs (after v. Jacksch).

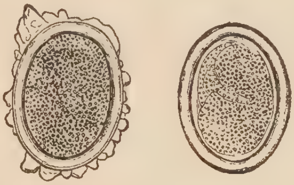

Fig. 339.—Eggs of Ascaris lumbricoides, double shell; albuminous envelope, magnified (Mosler and Peiper).

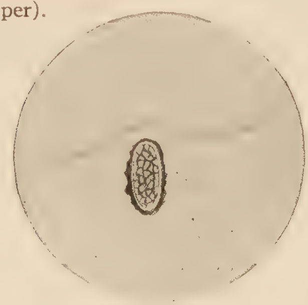

Fig. 340.—Unfertilized egg of Ascaris lumbricoides; ×500 (Logan).

Infection usually takes place by eggs in the soil near dwellings, in the drinking-water, and especially in raw foods, such as salads and fruits. These worms occur more frequently in children from three to twelve years of age and in the poorer class. They are not so frequent in adults. Females seem more frequently infected.

*Migration.*—The worms may crawl into the stomach, whence they may be ejected by vomiting; or they may pass through the esophagus and enter the pharynx, whence they may be withdrawn. The worm has entered the larynx and has produced fatal asphyxia, or into the trachea and lungs and caused gangrene. They have passed through the Eustachian tube and appeared at the external meatus. They have been found in the bile-ducts, pancreatic duct, the gall-bladder, and even in the liver, where they produced fatal abscess. They have entered hernial sacs, perforated intestinal ulcer, and some claim even the healthy bowel wall has been perforated by them. Appendicitis has been attributed to the ascaris; and obstruction of the bowel has been produced by a large mass of ascarides.

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[1] Twentieth Century Practice of Medicine, vol. viii, p. 583.

*Symptoms.*—They may produce no symptoms. In children, irritability, restlessness, picking at the nose, grinding the teeth, twitchings or convulsions, have been attributed to them. Anorexia, nausea, irregular bowel action, meteorism, irregular pulse, and black rings around the eyes may also occur. In rare instances progressive anemia has been observed. Itching of the nose may be present. The worms probably produce local hyperemia of the intestinal walls.

Hemorrhage from the bowel, simulating an occult ulcer, may be produced by a lumbricoid worm. The following interesting case is reported to me by H. D. Meeker. The possibility of hemorrhage from an occult duodenal ulcer was suggested. The author was present during part of the operation and examined the worm.

Mrs. L. P. consulted me in April, 1911, for irregular bleeding from the bowel. She gave the following history: Age twenty-five years, housewife, married four and a half years, no children, family history negative. She could recall no serious illness of any sort. Four years ago a slight bleeding from the bowel was noticed, a few weeks later a second hemorrhage occurred, and the patient purchased medicine for piles. There was no pain associated with the attacks. The hemorrhages continued at intervals of one to six weeks, the amount of blood varying from a mere trace to nearly a quart in twenty-four hours. The blood was always dark in color and usually clotted. The patient, after several weeks of treatment by her family physician, was sent to a Boston hospital where she remained a month without apparent benefit. She then went to Baltimore to live, where she received both private and hospital treatment. Not finding relief at the hands of the various internists, she sought help from the ray-enthusiasts and Christian Science with equally disappointing results. The day prior to admission to the Red Cross Hospital the patient had passed approximately 20 ounces of blood-clots. She was anemic, her blood count showing 3,500,000 red blood-corpuscles. Hemoglobin, 70 per cent. Aside from an indefinite tenderness of the abdomen, she gave no subjective symptoms, appetite and digestion had always been good, bowels inclined to be loose. Stomach washings and examination of the stools were negative in so far as throwing light on the cause of the condition was concerned. The sigmoidoscope revealed a healthy intestinal mucosa. In view of the negative, subjective and objective symptoms, and yet persistent bleeding, exploratory laparotomy was proposed and welcomed by the patient. A small right Battle incision was made in the mid-abdomen, permitting exploration in both directions. All the abdominal viscera were systematically examined and found with the exception of ileum as hereafter described. The small gut was examined inch by inch and appeared normal up to a point about 5 feet from the ileocecal valve where an area about 5 feet long was found deeply injected. At the mesenteric border the condition of the tissue suggested a healed ulcer. Another injected area was found 2 feet nearer the valve about 6 inches in length. Within this area doubled on itself, was felt a moving worm. The intestine was opened after observing the usual protective technic, and the worm extracted. A small bleeding was observed at the mesenteric attachment of the gut, this was excised, and the opening into the ileum closed. No other pathological condition of the abdominal contents was observed. The worm was of the Ascaris lumbricoid family, a male measuring 11¼ inches. The patient made a prompt recovery. She has been seen at intervals of two weeks and up to the present writing has had no further bleeding. Her hemoglobin has increased 10 per cent. A second blood count has not been taken. Eosinophilia was never present at any time nor were any ova or other intestinal parasites discovered. The various hemorrhages which occurred for a period of four years were evidently due to perforations of the gut vessels by a single male worm.

Chronic intestinal obstruction, gradually becoming acute, may be caused by a large mass of the ascaris lumbricoides. Such a case has been described under Acute Intestinal Obstruction.

*Diagnosis.*—This is made by the detection of the worm or of its ova in the stools.

*Treatment.*—The stools should be disinfected by carbolic (5 per cent.) or bichlorid (1 : 1000) solution to destroy the ova. The hands should be disinfected and all food protected against infection.

It is preferable to administer a simple saline cathartic for a couple of days and keep the patient on a light diet before administering the anthelmintic.

Santonin is the best remedy. It can be given mixed with sugar in doses of $\frac{1}{3}$ to $\frac{1}{2}$ grain (0.022–0.032) for a child, and 2 to 3 grains (0.13–0.194) for an adult, followed by calomel or a saline purge.

It can be administered in divided doses; thus, santonin, 1 grain (0.06), three or four times a day, followed by a purge; or santonin, $3\frac{1}{2}$ grains (0.2), with castor oil, 2 ounces (60.0); give 1 teaspoonful for a small child; 1 dessertspoonful for a larger child; 1 tablespoonful for an adult, two or three times daily (Einhorn).

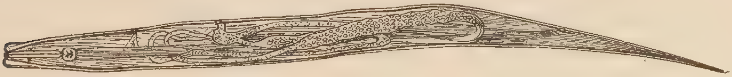

Fig. 341.—Oxyuris vermicularis: Female, enlarged (Mosler and Peiper).

Santonin, $\frac{1}{3}$ grain (0.022); hydrargyrum choridum mite, 1 to 2 grains (0.065–0.13). Give one powder t. i. d. Yellow vision (xanthopsia) occasionally follows the use of santonin.

*Chenopodium* (powdered seeds), in doses of 15 to 30 grains (1.0–2.0), or oleum chenopodii, 2 to 10 minims (0.118–0.592), followed by a cathartic, have been employed.

*Thymol,* $7\frac{1}{2}$ to 30 grains (0.5–2.0), given in capsules in divided doses and followed by a saline cathartic, has been recommended.

*Enteroclysis* with water, to which a few drops of benzine are added, has been suggested; but I scarcely see the benefit, as the habitat of the worms is in the small intestine.

**Ascaris Mystax.**—This is a round worm resembling the ascaris lumbricoides, but smaller and thinner. It is found chiefly in cats and rarely in man. No special symptoms result.

**Oxyuris Vermicularis (Thread-worm; Pin-worm; Seat-worm; Awl-tail; Maggot-worm).**—This parasite occupies the rectum and colon. It is white and filiform.

The male measures about 4 mm. ($\frac{1}{6}$ inch) in length and the female about 10 mm. ($\frac{2}{5}$ inch) (Figs. 341 and 342). It has three small nob-like lips. The female has two uteri, passing backward and forward from the end of the vagina. The opening of the latter is situated above the middle of the body. The eggs are 0.05 mm. long and about 0.02 mm. wide, with granular contents and white shell (Figs. 342 and 343).

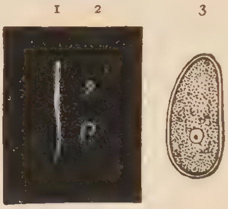

Fig. 342.—Oxyuris vermicularis, natural size: 1, Female; 2, males; 3, ovum, magnified (Vierordt).

Huber and others claim that they are generally deposited outside the body, so that feces rarely contain them; while Osler holds that they are usually found in the feces in large numbers. These worms occur at any age, though most commonly in children. The worms are found in the feces, at the anus, or in the vagina.

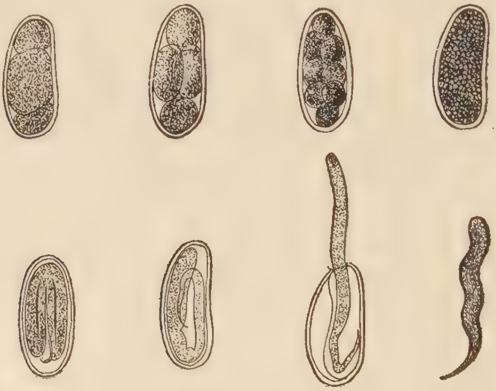

Fig. 343.—Segmentation and development of embryo of Oxyuris vermicularis (Heller).

Infection takes place through drinking-water or through salads, radishes, fruits, etc., the ova being dried upon them, or through the unwashed hands of the host.

The eggs of the oxyuris reach the stomach, when the shell opens and the embryo migrates into the small intestine. After fructification the females pass along the canal to the cecum, where they remain until the eggs are ripe, and then pass on downward, chiefly to the sigmoid and rectum. The oxyuris may traverse the intestinal wall and have been found in the peritoneal cavity, where they may form verminous tubercles in Douglas' fossa or perirectal abscesses.

*Symptoms.*—The oxyuris produce great irritation and itching around the anus, particularly at night. The pruritis ani is pronounced. The patient becomes nervous and irritable, sleep is disturbed. There may be anorexia, nausea, dizziness, palpitation, pollutions in the male, occasionally diarrhea, and at times anemia. Sometimes the parasites enter the vagina and cause irritation or nymphomania.

*Diagnosis.*—The worms are easily detected in the feces and are readily diagnosed by their appearance and location through inspection of the anus.

*Treatment.*—Cleanliness of the hands of the infected person and disinfection of stool, clothes, and bedclothes are important. Sleeping with an infected case should be forbidden.

Raw fruits should be cleaned and peeled, salads, etc., thoroughly washed. One should not eat out of the same vessels as the patient.

Santonin, administered by the same method as for ascaris, is an excellent remedy. Local treatment by enemata of water, 1 quart (liter) containing 5 to 10 minims (0.296–0.592) of benzine, or vinegar 1½ to 2 ounces (45.0–60.0), or thymol (1 : 2500) by enema, or fluidextract of quassia, 10 to 30 minims (0.292–1.704) to the quart; or soak a quassia cup in water for half an hour and inject quassia water, 1 pint (500 c.c.).

An enema of spirits (oil) of turpentine, 1 dram (4.0) to 1 pint (500 c.c.) of water, is of value.

These injections should be given with hips elevated and retained for a short period. Cold injections of strong salt water are of service in children. Carbolic acid I believe unsafe. Injections of lime-water are of use.

*Blackwash*—calomel, 1 dram (4.0); lime-water, 1 pint (500 c.c.)—locally, externally; lead-and-opium lotion, unguentum belladonna, vaselin, or

| | | |
|---|---|---|
| ℞. | Unguent. belladonnæ | ʒij (8.0); |
| | Tr. aconite radix | ʒss (2.0); |
| | Zinc oxid | gr. xv (1.0); |
| | Unguent. aq. rosæ . . . q. s. | ℥j (30.0).—M. |

Sig.—Ft. ung. External use to anus.

Cocain, ½ grain (0.32), can be added to this. These preparations lessen itching.

The saline enemata, given frequently, are of use.

**Ankylostoma (Anchylostoma) Duodenale; Uncinariasis; Hookworm Disease; Miner's Anemia; Egyptian Chlorosis; Dochmius Duodenalis or Strongylus Duodenalis.**—In 1843 Dubini first described this parasite in man. Griesinger, in 1854, demonstrated it as the cause of Egyptian chlorosis. Subsequently it was described in the tunnel workers of St. Gothard, and it is now recognized as an important cause of tropical anemia and of the anemia of miners, brick workers, and tunnel workers.

It is interesting to note that as far back as 1886, the late Professor Joseph Leidy reports in an article entitled "Remarks on Parasites and Scorpions," May 5, 1886, his discovery of true ankylostoma duodenale infesting a cat. He refers to the probability that some of the cases of supposed pernicious anemia found in America are due to this parasite. Leidy's researches are to be found at the Smithsonian Institution.

*Incidence.*—This parasite is widely spread in tropical and subtropical countries, and is one of the most fatal of parasitic diseases. In Porto Rico, in 1903, 5736 deaths out of a total of 23,433 were from anemia due to uncinariasis, as shown by the Anemia Commission in the report issued by Ashford, King ~~and~~ Igaravidez. A summary of ten years' campaign against hookworm disease is reported by these authors.[1] Stiles has demonstrated that the disease is endemic in many places, and is the cause of the common anemia in the Southern States.

Harlow Brooks[2] reports a case of hookworm infection endemic in New York. This patient had been working on the reservoir at Brewster, N. Y. He was of Irish-American extraction. A number of his associates were Italians recently arrived in this country. The European form of the worm was found in this patient's stools. Stiles[3] has examined the pupils of eight schools, 1306 children inspected, and classified 55.9 per cent. as "suspects" as regards hookworm disease in Southern Florida. He believes the condition lamentably common in the South. Wells[4] investigating uncinariasis in the State of Georgia, finds, *exclusive of the large towns*, about 24 per cent. of the total white population have uncinariasis. Infection seemed to be confined chiefly to those between five and twenty-seven years of age, and the parasites may live in the intestines from two to twelve years.

Bass[5] has reported a large number of cases in the country population of Mississippi. Uncinariasis has been found among the miners in Pennsylvania.

Gunn[6] reported in 1905 about 60 cases of imported hookworm disease in the state of California. Sprague and Endicott have reported the occurrence of this disease in Jackson, Amador Co., in the same state, and Gunn[7] more recently reports a large series of cases of hookworm disease in the mines of California, and on investigation finds the disease is endemic in certain mines, hookworm embryos being demonstrated in the earth removed from these mines. The condition is also found in Nevada and probably exists in many other mines in the United States. Unsanitary conditions in the mines are undoubtedly responsible. In many of them there are no privy facilities underground. Protection against this infection is of interest on the economic as well as on the humanitarian side. Ford[1] reports a case of hookworm disease in Kansas.

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[1] Jour. Amer. Med. Assoc., May 28, 1910.

[2] Med. Rec., Jan. 29, 1910.

[3] Public Health Reports, March 25, 1910.

[4] Jour. Amer. Med. Assoc., June 4, 1910.

[5] Ibid., July 21, 1906.

[6] California State Jour. Med., April and Aug., 1905.

[7] Jour. Amer. Med. Assoc., Jan. 28, 1910.

It is not uncommon in the Philippines. Chamberlain[2] reports a statistical study of hookworm among the whites in the Philippines, and Castellani and Chalmers in the July issue of the same Journal, intestinal flagellates occurring with hookworm. The disease is prevalent among the miners of Germany and Austro-Hungary and also in Westphalia. The anemia of the Cornish miners has been shown to be due to hookworm. In Egypt the disease is very prevalent.

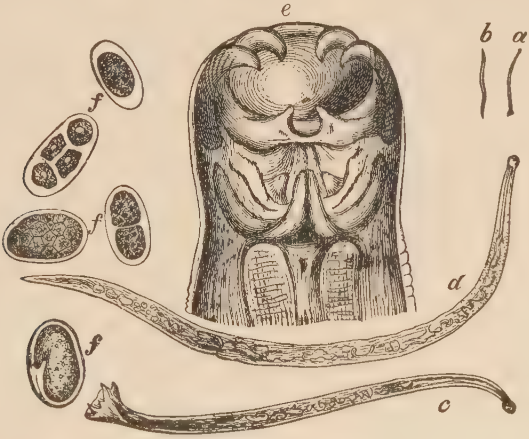

Fig. 344.—Ankylostomum duodenale: *a*, Male (natural size); *b*, female (natural size); *c*, male (enlarged); *d*, female (enlarged); *e*, head; *f*, eggs (after v. Jaksch).

*Parasite.*—The worm is a strongyle, closely allied to the sclerostoma, which causes verminous aneurysms and colic in the horse, and to the gapeworm of fowls. There are two forms—the Anchylostoma duodenale and the Uncinaria americana or Necator americanus—described by Stiles. They have the same general characteristics, there being certain differences in the arrangement of the teeth, etc.

The worm is cylindric in shape, about 0.5 to 1 mm. thick. The males are from 7 to 11 mm. in length, the females 10 to 18 mm. The American worm is the longer. The worm is yellowish or grayish-white in color, with translucent edges. The head is curved toward the dorsal surface and the mouth is provided with a heavy armature of hook-like teeth, with which they pierce the mucosa. There is a strong muscular esophagus. The male has a prominent caudal expansion or bursa (Fig. 344).

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[1] Jour. Amer. Med. Assoc., May 28, 1910.

[2] Philippine Jour. of Sci., Aug., 1910.

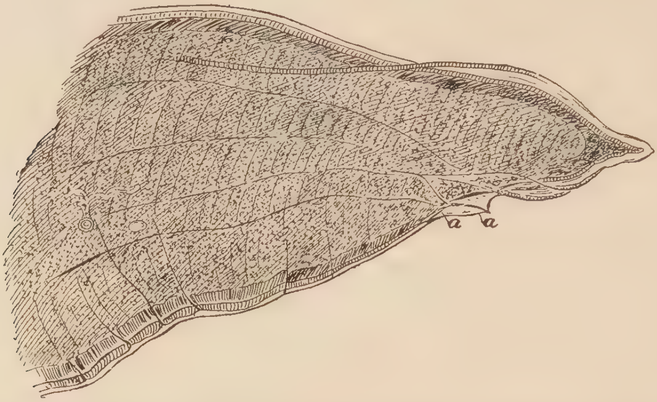

Fig. 345.—Posterior extremity of female *Uncinaria Americana*, viewed ventrolaterally, showing anal opening expanded: *a, a,* Anal papillæ, showing small chitinous tips (A. J. Smith).

In the female the caudal end is pointed and armed with an awl-like prong (Fig. 345). The female worms number about four to one. The eggs are oval, 64 to 76 $\mu$ long by 36 $\mu$ wide (Fig. 346) in the American form. They are laid in segmentation. The development is direct, without an intermediate host. The European eggs are smaller. Sometimes as many as 2,000,000 eggs are found in one stool. They do not hatch in the intestines. Sometimes they are not readily found in the microscopic field and centrifuging the specimen may be necessary.

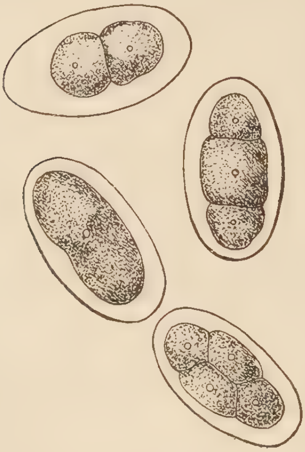

Fig. 346.—Four eggs of the New World hook-worm (*Uncinaria Americana*), in the one-, two-, and four-cell stages. The egg showing three cells is a lateral view of a four-cell stage. These eggs are found in the feces of patients, and give a positive diagnosis of infection. Greatly enlarged (after Stiles).

Bass affirms that more than 7 per cent of females out of 247 whom he dissected failed to produce ova. He maintains that during the latter third of their existence, they cease to lay eggs. If this observation is substantiated, in some cases individuals may be harboring the worm, though no ova can be found in their stools.

The embryo lives in the water or moist ground and passes through the rhabditiform stage. Larvæ may live for months in the mud and water of the mines. They may be taken into the body by drinking-water, with the dirt from the hands of the miners and tunnel workers, or in the soil eaten by the earth feeders, the geophagi. They may be carried in the dust and contaminate green vegetables and fruit.

**Uncinarial Dermatitis.**—Ashford and King[1] refer to the fact that in Porto Rico infections through the mouth are rare, and that fully 96 per cent. of the patients have suffered from ground-itch (" Mazamorra "), or dew-itch, *due to invasion of the skin*[2] *by these larvæ. The well shod were never affected.* Various stages of dermatitis occur, and even obstinate ulcers of the leg, in the lower third especially. These were called " tropical " or " syphilitic " by the ignorant. Next in frequency to infection through the skin of the feet are the hands and arms as the route of entrance. Loos demonstrated that embryo worms enter the skin, the lymph-channels, and then the veins, and are carried by the veins to the right side of the heart and lungs, and then pass up through the trachea into the pharynx and are swallowed. Ashford further believes that skin infections can take place without manifest dermatitis from experiments on animals. Skin infection is probably very frequent. Infection is more common in summer than winter, in part probably due to the fact that greater protection is afforded the lower limbs during the cold or rainy season.

*Immunity.*—Some whites, and *especially the negro race* and Asiatics, enjoy considerable immunity to this disease. One may find a large number of ova in the stools, and yet there may be few or no symptoms. Usually the greater the infection, the more acute the onset and course of the disease. The *adult worm lives in the small intestine;* more *are found in the jejunum;* many in the duodenum; and rarely in the ileum or colon.

*Symptoms.*—In the early stage there may be gastro-intestinal symptoms, such as pains in the epigastrium (gastralgia), tenderness in the right hypochondrium, nausea, occasionally vomiting, constipation, rarely diarrhea. Pain in the sternum and chest, slight breathlessness on exertion, and palpitation. A temperature of 37.5° to 38.5° C. (99.5°–103.5° F.) is not uncommon. The patient feels unable to work; gradually anemia becomes manifest, the fever disappears, hemoglobin steadily diminishes. Headache, vertigo, tinnitus aurium, hemic murmurs, weakening of the pulse, and dyspnea follow. The pains in the sternum become quite severe, debility and mental hebetude increase; patellar reflexes are diminished or even lost. Impotence or amenorrhea may be present.

In this advanced condition the skin is of a dirty muddy hue, at times waxy white. In the Southern States it is known as the " Florida complexion." The eyes are dull, heavy, lack lustre, and have a blank stare. Children are interfered with in their growth and become stunted and ill developed. The circulatory system becomes more profoundly affected; the heart, at first slightly hypertrophied, now becomes dilated and broken compensation ensues. There are edema of the feet and legs, puffiness of the face and a general anasarca, with pericardial, pleural, and peritoneal effusions. The patient is bedridden and gradually passes away in a semisomnolent condition. Acute cardiac dilatation, cerebral effusion, or violent diarrhea may be terminal events.

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[1] Uncinariasis, Jour. Amer. Med. Assoc., Aug. 10, 1907.

[2] The skin eruption known as " bunches," occurring in the Cornish miners is probably due to the entrance of these worms (Haldane).

Osler holds that the liver and spleen become enlarged, but this does not correspond to the findings of Ashford and King. The hemoglobin may fall below 30 per cent. and sometimes even to 8 per cent., and some of these latter have recovered under appropriate treatment. Eskridge[1] reports a case of Ainhum complicating uncinariasis.

*Morbid Anatomy.—Autopsies* (Ashford and King).—Muscles are often brownish gray, friable and atrophied. Skin and subcutaneous tissue pale and sodden with fluid. Serous effusion generally present in the pleuræ and pericardium, sometimes in the cerebral ventricles. Ascites marked.

*Lungs.*—Edema and passive congestion.

*Liver.*—Never normal; once was increased in size; never diminished; fatty degeneration often present; connective-tissue increase is not a feature.

*Kidneys.*—Chronic parenchymatous or chronic diffuse nephritis. As a rule, there was little connective-tissue increase.

*Stomach.*—Uncinariæ are occasionally found in the stomach and even adherent to its walls. Chronic gastritis is common.

*Intestines.*—The jejunum contained most of the uncinariæ, some unattached and others attached to the mucosa. The balance were mostly found in the first part of the duodenum, a few in the ileum, and none in the colon.

The intestinal lesion is confined to the mucosa, there *being a tiny superficial erosion* about 0.5 mm. ($\frac{1}{50}$ inch) in diameter, and not a deep ulcer. They are usually not red and are difficult to find, except with a hand lens. The duodenum and especially the jejunum are the seat of a chronic intestinal catarrh.

The dejecta are often of brownish color. Microscopically, eggs of the parasites and at times Charcot-Leyden's crystals are found in the stools.

*Spleen.*—This is frequently found reduced in size, soft, and has a wrinkled capsule. There was a paucity of lymphoid elements.

*Hemolymph Glands.*—In the region of the abdominal aorta, especially near the bifurcation, enlarged glands of dull reddish hue were noted. There was no surrounding trace of inflammation. Microscopic examination showed they were hemolymph glands of the type described as splenolymph.

*Bone-marrow.*—Changes such as occur in pernicious anemia; also eosinophilous cells.

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[1] Med. Rec., Sept. 17, 1910.

*Anemia.—Blood.*—The findings vary from a diminution of hemoglobin and red cells to those of *pernicious anemia*. The hemoglobin is usually relatively lower in uncinariasis; as low as 15 to 20 per cent. in severe cases is not uncommon, 8 per cent. having been registered.

Polychromatophilia, and in severe cases poikilocytosis, with macrocytes and microcytes, occur. Normoblasts and megaloblasts were not uncommon, but the latter were in the minority. Erythrocytes averaged 2,406,416 per cubic millimeter, the lowest 754,000; average hemoglobin slightly over 40 per cent.

Osler reports marked leukocytosis in his cases. Some observers hold that leukocytosis is generally due to complications; while Ashford and King find no constant leukocytosis, but that it is present at times in acute cases, while in chronic cases there is apt to be leukopenia. There seemed to be a tendency of the leukocytes to degenerate.

Eosinophilia is of importance. Boycott and Haldane hold it is present in 94 per cent. of cases, and at times it is quite high. Ashford and King call especial attention to the fact *that in the most serious cases it is liable to be absent*, and that it is chiefly of prognostic importance. Very chronic cases of severe type, poor resisting power, and lack of blood regeneration, they state, rarely show eosinophilia, or only to a slight degree; that a rise of eosinophilia is of good prognostic significance; that a fall, with lack of improvement in the symptoms, is not a good omen. They hold that good resistance to the toxin of uncinariæ is expressed by eosinophilia. An interesting case of fatal acute hook-worm infection with purpura is reported by Bolling Lee and Harlow Brooks.[1] Eosinophilia was absent probably by reason of the severe character of the infection and the slight body resistance to it.

*Diagnosis.*—By the finding of the eggs and parasites in the stool. It is well to examine the stools after a dose of thymol, followed by a saline cathartic. Stiles suggests placing a small bit of feces on a white blotting paper when a microscopic examination cannot be made. In about an hour there is a blood-red or reddish-brown stain suggestive of blood. The worms to the naked eye are about ½ inch long, of the diameter of a pin, with one end sharply recurved.

Eosinophilia is suggestive. The microscopic examination of the stool will settle the diagnosis. It is interesting to note that *other intestinal parasites or their ova* may be found in addition to the hookworm. Charcot-Leyden's crystals in the stool are also suggestive.

*Prognosis.*—This is good if the condition is detected early, but is bad in the advanced cases.

*Prophylaxis.*—In camps or mines, proper sanitary regulations, such as the correct location of the latrines, etc., are important. In regions where this infection is endemic, new miners should be inspected, and infected cases should be pronounced free from the disease before being allowed to resume work. The feet and legs should be protected from the soil (should not be bare) and the hands properly scrubbed before eating. The stools of infected patients should be disinfected with bichlorid of mercury (1 : 1000) or carbolic acid (1 : 20) solution.

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[1] Med. Rec., May 14, 1910

Raw fruits and vegetables, such as apples, lettuce, etc., should be properly cleaned, and should preferably be avoided if there are many cases of ankylostomiasis.

*Treatment.*—Filix mas, thymol, and betanaphtol are the best drugs. The ethereal extract of filix mas, same dosage as for tapeworms, and then the solid extract were tried in Porto Rico, but they seemed to be of no value. Ashford believes that possibly the preparation deteriorates in warm climates. In the early stage of emigration (uncinarial dermatitis) the author suggests the possible value of urotropin, 10 grains (0.6), with same quantity of sodium benzoate, four to six times a day.

*Thymol proved to be the best remedy.*—The day previous the patient abstains from solid food and at night is given 25.0 grams sodium sulphate or any good saline cathartic; the next morning, if free bowel action, on an average about 30 grains (2.0) of thymol should be given in capsules, and an equal dose two hours later, followed in a couple of hours by another saline cathartic.

This procedure should be kept up once a week, until no ova are found. Smaller, occasionally even larger, or more frequent doses may be required. It is generally advised that oily cathartics, such as castor oil and also alcohol, should not be taken directly after the thymol. Dizziness or slight collapse may follow the use of thymol. Epigastric pain, nausea, vomiting, tremors, twitchings, convulsions, and severe collapse, pulmonary congestion and fatty degeneration of the liver result from thymol poisoning. Ashford mentions cases who have taken alcohol for these conditions with no deleterious effect.

Occasionally thymol, when given pulverized in capsules, may pass through in a hard stony mass, with no effect on the parasites. Lindeman[1] recommends triturating the thymol crystals with equal parts of milk-sugar and placing the mixture in cachets which are softened to the consistency of a raw oyster before administering. The sugar of milk is dissolved and the thymol is well distributed over the small intestine. Lindeman believes that intestinal catarrh, rapid peristalsis from intestinal erosions, and especially dilatation of the stomach, with gastroptosis, *which latter condition he believes frequent*, as many of these patients are pot-bellied, that these conditions interfere with the thymol coming in contact with the parasite. With ectasy, the drug would lie too long in the stomach and part be absorbed. It is, therefore, recommended that the patient lie on the right side after ingestion of the thymol, so the stomach can readily empty itself. The following dosage is outlined by the State Board of Health of Florida.

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[1] Jour. Amer. Med. Assoc., May 28, 1910

| | |
|---|---|
| Under 5 years of age | Up to 8 grains. |
| 5 to 10 years of age | 8 to 15 grains. |
| 10 to 15 years of age | 15 to 30 grains. |
| 15 to 20 years of age | 30 to 45 grains. |
| 20 to 60 years of age | 45 to 60 grains. |
| Over 60 years of age | 45 grains. |

One should also consider the weight of the child, and also in all cases the condition of the heart and the degree of debility.

Betanaphtol, 15 grains (1.0), and two hours later the same dosage, administered like thymol, have been employed. It is somewhat irritating to the kidneys; is an excellent anthelmintic, but not quite as safe as thymol nor as efficacious.

Ashford and King hold that hemoglobin increased more rapidly under the use of iron, but that the return to normal came about as surely without it, after the use of thymol. They used Blaud's pills in obstinate cases. Iron and arsenic I believe valuable accessories.

℞. Blaud's pill (iron) (made fresh) ........... gr. v (0.3);
Sod. arsen ........................... gr. $\frac{1}{50}$ (0.0013).—M.
One pill.
Sig.—One t. i. d.

Peptomangan (Gude), 1 to 3 drams (4.0–12.0), or Blaud's iron pill, 5 to 10 grains (0.3–0.6), or iron tropon can be used, combined with Fowler's solution of arsenic, 5 minims (0.274), t. i. d.; or atoxyl, $\frac{1}{3}$ grain (0.02), by hypodermic every other day.

A subsequent high enema of thymol, 1 quart (liter), 1:2500, might be of value in aiding their destruction after the worms have passed into the large intestine.

**Strongyloides Intestinalis.**—Under this name we now include the small nematode worms found in the feces and formerly described as Anguillata stercoralis, Anguillata intestinalis, and Rhabdonema intestinale. The parasite occurs abundantly in the stools of the endemic diarrhea of hot countries, and has been described by the French in the diarrhea of Cochin-China. It has been found in Manila by Strong.

W. S. Thayer reported 3 cases from Osler's clinic. It has occurred in Italy.

Blumgart[1] reports their larvæ in a case and refers to reports of 5 additional cases, and to the fact that Southern physicians refer to other cases, so that the disease is probably more widespread than is supposed. The worms are said to occupy all parts of the intestines, and have even been found in the biliary and pancreatic ducts.

The female is from 1 to 2.20 mm. long and 0.04 to 0.03 mm. wide; the male is about a fifth smaller (Fig. 347). The mouth has three distinct lips, continuous with a triangular esophagus, which after narrowing dilates again into a second ovoid enlargement, which is followed by intestines. The intestinal tract is bordered by fine granulations.

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[1] Med. Rec., April 6, 1907.

Eggs are elliptic, with a thin, clear yellow shell, with granular contents about 0.00675 by 0.0375 mm. They hatch quickly, so are rare in the stools. They occur chiefly in the duodenum and jejunum, but have been found in the stomach and other parts of the intestines.

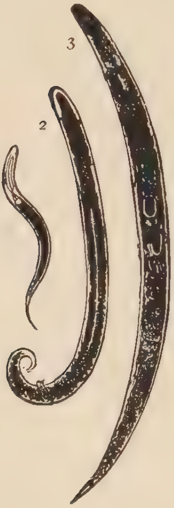

Fig. 347.—Strongyloides intestinalis and stercoralis: 1, Larva (Anguillula intestinalis); 2, male Anguillula stercoralis; 3, female Anguillula stercoralis (after Perroncito).

*Symptoms.*—They are usually more of chronic diarrhea than of dysentery. There are at first mild dyspeptic symptoms, eructations, anorexia, and a diarrhea of moderate intensity, with soft and pasty stools, three or four a day, often in the early morning hours. The attacks are sometimes dysenteric, with mucus and blood; in other cases they are more choleraic, the dejecta consisting of liquid yellow material; while vomiting, cyanosis, and collapse may occur.

Emaciation and prostration may be present. Anemia is, as a rule, not very severe. Intercurrent dysentery is not uncommon, also headache, vertigo, tinnitus, aurium, and prostration.

*Treatment.* — Rest and liquid diet. Male fern, ethereal extract, 12 to 30 grams divided in three doses during the morning and repeated daily, have been used by the Italians.

*Thymol has been quite successful.*

Large quantities of olive oil have seemed to give good results in some cases.

**Trichocephalus Dispar (Whip-worm).**—This parasite is found in the cecum and large intestine of man. It measures from 4 to 5 cm.

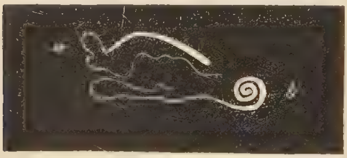

Fig. 348.—Trichocephalus dispar: *a*, Female; *b*, male (natural size) (Heller).

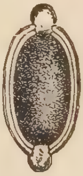

Fig. 349.—Egg of Trichocephalus dispar, moderately enlarged (Heller).

long, the male being smaller than the female. It is readily recognized by the peculiar differences between the anterior and posterior portions. The anterior forms three-fifths of the body, is thin and hair-like; the tail end of the female is more conic and thicker, terminating in a blunt extremity; while that of the male is rolled like a spring (Fig. 348).

The ovum is lemon-shaped, dark brown, 0.05 mm. in length, and provided with button-like projections (Fig. 349).

The number of worms is variable, as many as a thousand having been counted; often only ten to twenty are found. In parts of Europe they are very common, but not so in the United States. Profound anemia with diarrhea have occurred from them. Many worms may be present without producing symptoms; occasionally diarrhea and nervous symptoms occur.

*Diagnosis.*—This can be made from the peculiar ova. Living worms are rare in the stool.

*Treatment.*—Thymol, as previously described. Extract of male fern may be employed. High enemata of warm water, 1 quart (liter), containing 5 to 10 drops of benzin, may be of service.

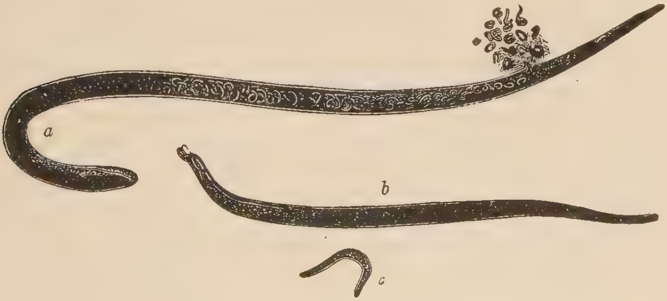

Fig. 350.—Trichina spiralis (greatly enlarged): *a*, Female; *b*, male; *c*, embryo (after Heller).

**Trichina Spiralis (Trichiniasis).**—The trichina in its adult condition lives in the small intestine. The embryos pass from the intestines and reach the voluntary muscles, where they become encapsulated larvæ.

*Muscle Trichinæ.*—Tiedemann, in 1822, described the ovoid cysts in human muscle. Owen named the parasite. Leidy, in 1845, described it in the pig. In 1860, Zenker discovered in a girl both the intestinal and muscle forms, and established their connection with the specific symptoms.

*Incidence.*—Man is infected by eating the raw or not completely cooked flesh of trichinous hogs, which contain encapsulated trichinæ. The capsules are digested in the stomach and the trichinæ set free. They pass into the small intestine, and about the third day become sexually mature. On the sixth or seventh day the embryos are fully developed. The young produced by each trichina (female) have been estimated at several hundred. The female worm penetrates the intestinal wall and the embryos are probably discharged directly into the lymph-spaces, and thence into the venous system, whence they reach the muscles; and in about two weeks they develop into the full-grown muscle form. A myositis is produced and they may become encapsulated. The trichinæ may live therein for many years. Calcification may occur about them. One must remember that in the hog the capsule does not readily become calcified, so that the parasites are not as readily visible as in man. Moreover, an apparently healthy looking animal may be suffering from trichiniasis.

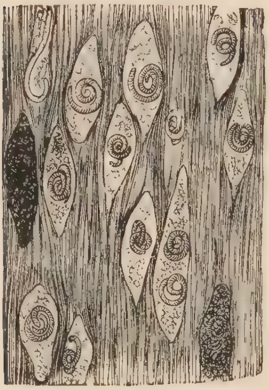

Fig. 351.—Fresh muscle trichinæ (Mosler and Peiper).

*The intestinal trichinæ are visible to the naked eye*—white glistening worms 3 to 4 mm. long; and the male half this size, with two little projections from the hind end. The caudal extremity is thicker than the head (Fig. 350).

The muscle trichina is 0.6 to 1 mm. long and coiled in the capsule. It has a pointed head and rounded tail (Fig. 351).

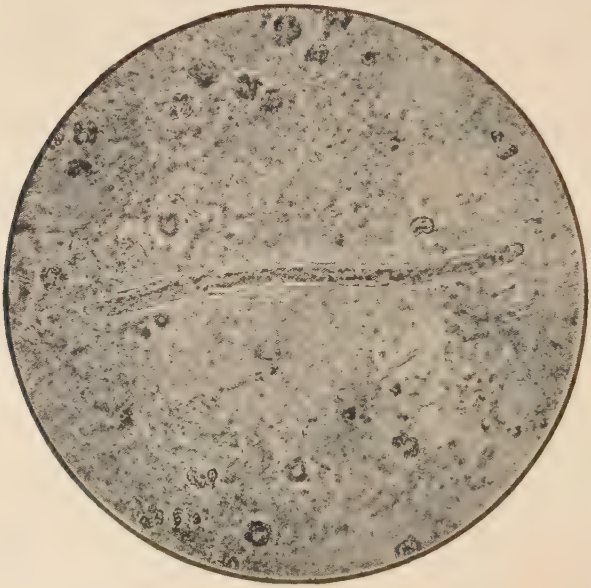

Fig. 352.—Trichinella spiralis in blood (×500) (Packard).

**Trichinella Spiralis in the Human Blood.**—Herrick and T. Janeway[1] have demonstrated the Trichinella spiralis in the human blood, and E. Packard[1] reports finding an embryo of the Trichinella spiralis in the blood of a patient and within a short period larger embryos in the muscle, not yet encysted (Figs. 352, 353).

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[1] Arch. Int. Med., 1909, iii, 263.

*Symptoms.*—As a rule, a few days after eating the infected flesh gastro-intestinal disturbances occur, such as pain in the abdomen, anorexia, vomiting, and at times diarrhea. The attack may resemble cholera nostras or even typhoid fever. Invasion symptoms usually occur between the seventh and tenth days or, occasionally, not until the end of two weeks. There may be fever, intermittent or remittent, even to 102° to 104° F. Chills are not common.

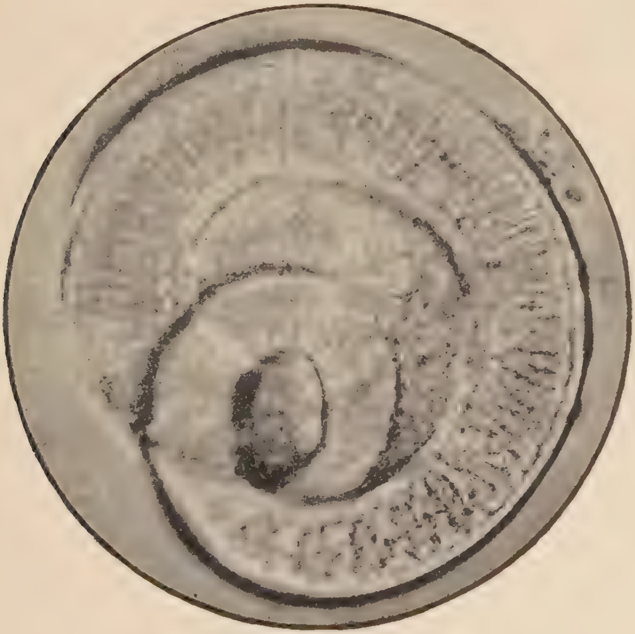

Fig. 353.—Trichinella spiralis found in muscle five days after embryo was found in blood (× about 250) (Packard).

Pain occurs in the muscles on pressure and movement, also swelling of the muscles. There may be difficulty in chewing and swallowing. Respiration may be painful. Dyspnea may be present from involvement of the intercostal muscles and diaphragm. *Edema*, especially *about the eyes*, is an important symptom. Sweating, itching, and urticaria occur. The general nutrition is disturbed and the patient becomes anemic and emaciated. In cases with severe infection, there may be delirium, tremor, and dry tongue, suggestive of typhoid fever. Bronchitis, pleurisy, pneumonia, polyuria, or albuminuria may occur.

*Diagnosis.*—Trichinosis has been mistaken for typhoid fever and for articular or muscular rheumatism, multiple neuritis, or cholera morbus. With typhoid there is leukopenia, the presence of the Widal reaction, and the typhoid temperature. The typhoid bacilli are also present in the blood and stools.

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[1] Jour. Amer. Med. Assoc., April 16, 1910.

Leukocytosis, *especially marked eosinophilia*, is an aid to the diagnosis of trichinosi. Charcot-Leyden crystals occur in the stools.

Eosinophilia, edema of the eyelids, dyspnea, swelling, and tension of the muscles are at once suggestive. The presence of trichinæ in the stools and muscles is conclusive. The muscle can be incised under cocain injection and the cyst examined.

The disease has proved fatal in a number of cases.

*Prognosis.*—This depends on the *intensity of the infection.* Mild cases may recover in two weeks. The mortality has ranged as high as 30 per cent. Early diarrhea is favorable to evacuate the infected pork.

*Prophylaxis.*—Pork, such as ham, sausage, etc., should always be thoroughly cooked before eating. Proper inspection of hogs is important.

*Treatment.*—Immediate lavage, if infected pork is suspected and the case is seen early. Evacuation of the bowel by calomel, 10 grains (0.6), or a saline cathartic. Thymol, santonin, male fern, kamala, and turpentine, have all been recommended as vermifuges in the early stage. Glycerin in large doses internally is said to be destructive of the worm. Later, the treatment is symptomatic and the strength is to be supported. Urotropin, in 10-grain (0.6) doses four to six times a day, I believe of value during the early stages. It is preferable to combine it with equal doses of sodium benzoate.

The writer has secured an excellent result by the use of urotropin in a recent case.

Picric acid, ½ to 2 grains (0.03–0.1) dissolved in alcoholic solution and well diluted, has been recommended to destroy the larval forms circulating in the blood. It is very bitter and liable to cause gastro-intestinal irritation. Quinin has also been recommended. I do not advise their use.

# PART IV

# DISEASES OF THE PANCREAS

## CHAPTER XXXV

## THE PANCREAS AND ITS ANOMALIES—SURGICAL RELATIONS

THE greater part of the pancreas is located in the epigastrium (Fig. 354), but a portion of the body and tail extend into the left hypochondrium, and the head may project into the umbilical region. The organ lies about 3 inches above the umbilicus, midway between

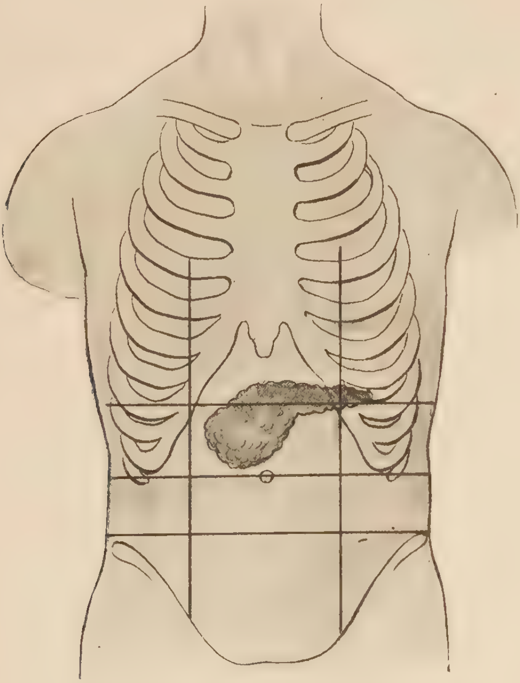

Fig. 354.—Surface marking of the pancreas.

the navel and ensiform appendix, the body corresponding to the level of the first lumbar vertebra.

To expose the pancreas one must detach the stomach from the great omentum and turn it upward. The pancreas is a long, pink, cream-colored gland, which stretches transversely across the posterior abdominal wall from the concavity of the duodenum to the lower and inner border of the spleen (Fig. 355).

When fresh, its consistency is firm and its appearance is lobulated. It varies in length from 5 to 6 inches (12 to 15 cm.). Its weight averages from 2.25 to 3.5 ounces (66 to 102 gm.).

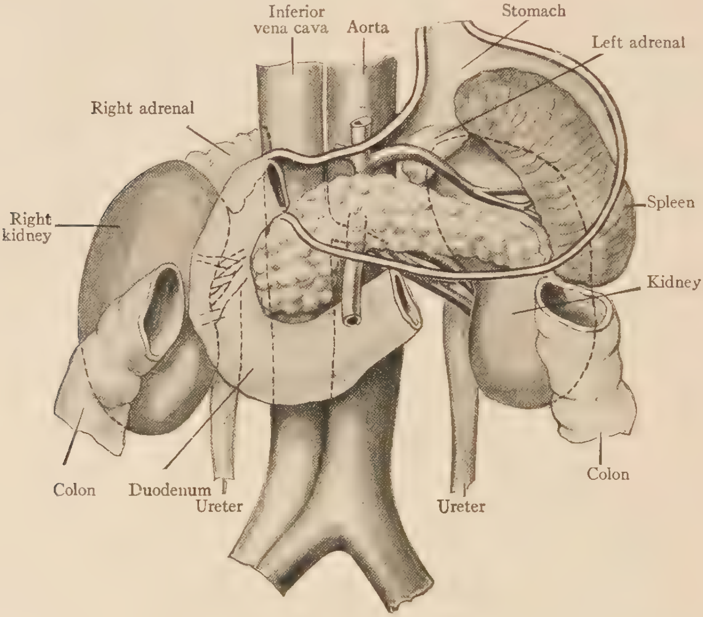

Fig. 355.—Diagram showing the relations of the pancreas (Robson and Cammidge).

The enlarged right extremity, or "head," extends downward and to the left, and lies in the concavity of the duodenum, in contact with its second and third parts. This portion is enlarged, bulbous, and lies opposite the second and upper part of the third lumbar vertebra. The short constricted part, or "neck," arises from the upper and anterior part of the head. It runs upward and slightly forward, and then to the left for about an inch, where it merges into the body of the gland. This last, which is the longest section, runs backward and to the left at the level of the first lumbar vertebra. The pointed left extremity ("tail") is least firmly attached, and it merges gradually into the body.

To the left of the head, in contact with the neck, is a deep groove, the "incisura pancreatis," in which lie the superior mesenteric vessels; a short process of the head, the "uncinate," projects behind and to the left of the vessels along the third part of the duodenum, deepening the groove in which they lie. Occasionally this process lies separate and is known as the "lesser pancreas."

The anterior aspect of the head is in contact with the commencement of the transverse colon. The liver overlaps the pancreas. The lower part of the head anteriorly is covered by peritoneum reflected from the lower surface of the colon, and which enters into the formation of the greater sac of the peritoneum. This part is in contact with the small intestine.

The posterior surface of the head has no peritoneal coat, and lies in front of the inferior vena cava, the left renal vein, and the aorta. The common bile-duct is in a groove or canal on this surface.

Anteriorly, and to the right, the neck is in contact with the first part of the duodenum, and with the pylorus when the stomach is distended. Behind, and to the left, there is a groove in which are the terminations of the superior mesenteric veins and splenic veins.

The **body** has three surfaces. Its anterior surface is concave, looking upward and forward. The lesser sac of the peritoneum separates it from the stomach. Where the body joins the neck there is often a prominence—the "omental tuberosity"—so called from its contact with the small omentum when the stomach is distended. The posterior surface looks backward and lies upon the aorta, the origin of the superior mesenteric artery, the pillars of the diaphragm, the splenic artery and vein, the left kidney and renal vessels, and the left suprarenal capsule. The splenic vessels run along its upper border in a single groove or, at times, in separate grooves. The posterior surface has no peritoneal covering, and is connected to the abdominal wall and adjacent organs by areolar tissue. The inferior surface looks downward and slightly forward. At the right end it rests on the *duodenojejunal* flexure. The middle part is covered by jejunum. This surface is invested by peritoneum derived from the descending layer of the transverse mesocolon. With gastroptosis, the pancreas may at times be felt in a space between the edge of the liver and the lesser curvature of the stomach.

The **tail** is in contact with the lower part of the inner surface of the spleen, but occasionally a portion of the mesentery, containing a lymph-nodule, intervenes.

**Blood-supply.—Arteries.**—The hepatic branch of the celiac axis, the inferior pancreaticoduodenal branch of the superior mesenteric, the superior pancreaticoduodenal artery (a branch of the gastroduodenal), and branches of the splenic and of the hepatic arteries supply the pancreas.

**Veins.**—These are tributaries of the splenic and superior mesenteric veins, the anterosuperior pancreaticoduodenal vein, which enters the superior mesenteric vein; the posterior pancreaticoduodenal vein, which empties into the portal vein; a number of small tributaries enter the splenic vein, and small veins from the head and neck, which run into the portal vein.

**Lymphatics.**—This system enters into glands located on the head of the pancreas, in the hilum of the spleen, and along the superior mesenteric vessels.

**Nerves.**—The vagi supply the cerebrospinal fibers, and the solar plexus the sympathetic fibers. They accompany the arteries through the celiac, splenic, and superior mesenteric plexuses, traverse the substance of the gland in company with the ducts, and terminate in plexuses around the acini, sending fibers to the secreting cells. The nerve-fibers are chiefly non-medullated, and have minute visceral sympathetic ganglia. Small nerve-cells are also found near the distribution of the nerve-fibers to the alveoli.

**Peritoneum.**—The transverse mesocolon is attached to the anterior border of the pancreas, from the neck to the tail. The anterior layer passes upward and backward over the superior surface, forming the posterior wall of the lesser peritoneal cavity. The posterior layer passes downward and backward along the inferior surface to form the greater peritoneal sac. At the neck and on the head anteriorly the two layers of peritoneum have separate lines of attachment, so that there is a space devoid of peritoneal covering, which is separated from the colon by areolar tissue. In some cases the transverse mesocolon is continued as far as the hepatic flexure, in which event the head and neck are completely invested. The posterior surface of the pancreas is uncovered by peritoneum.

**Ducts of the Pancreas.**—The pancreas has two ducts opening separately into the duodenum. The duct of Wirsung, the chief one, commences in the tail from the union of the small branches and gradually increases in size, passing through the body of the gland from left to right. It passes downward and backward through the neck into the head of the organ, where it lies nearer the posterior surface, and is in relation with the common bile-duct, beside which it runs into the duodenum. The two ducts pierce the second part of the duodenum obliquely, about 3 to 4 inches (8 to 12 cm.) below the pylorus, and afterward unite to form a common channel, the "ampulla" or "diverticulum of Vater," which opens by an orifice into the gut. This last is situated on a papilla-like fold of the mucous membrane, called the "papilla or caruncula major." Above it there is a small fold of mucous membrane, which must be raised in order that the orifice may be seen. Running downward from the caruncle there is a small vertical fold known as the "frenum carunculæ" or "plica longitudinalis."

The diverticulum of Vater is oval or triangular, having the duodenal orifice at its apex, and its base at the openings of the two ducts. Its average length is only 3.9 mm., though Testut believes it to be longer, from 6 to 7 mm., and rarely as much as 11 mm. The orifice of the diverticulum of Vater is the narrowest part of the biliary channel, only averaging 2.5 mm. According to Oddi, a thin layer of unstriped muscle-fiber surrounds the ampulla and the terminations of the ducts, forming a sphincter.

The duct of Santorini (ductus pancreaticus accessorius) drains a large part of the head of the pancreas. It anastomoses near the neck with the duct of Wirsung, is apparently a branch of this larger duct, and usually increases in size as it approaches it. The duct of Santorini diminishes in size as it approaches the duodenum and opens into the latter on a small papilla, the "caruncula minor," ¾ to 1 inch above and ventral to the "papilla major." Opie finds, in more than half the cases, that the orifice of the duct of Santorini is obliterated, or so constructed that it cannot assume the function of the larger duct when the latter is occluded. The main and accessory ducts communicate with each other by branches of varying size.

## ANATOMIC ANOMALIES OF THE PANCREAS

Depending upon alterations of the usual process of development various anomalies may occur in the pancreas. The tail of the pancreas has occasionally been bifid.

**Pancreas divisum** is due to an absence of lobules of parenchyma about the duct of Wirsung or its branches for a short distance, so that a division of the gland into two parts may result.

**Pancreas Minus.**—There is a supernumerary lobule or lobe present in the head of the gland, separated by a constriction. It cannot be classed as an accessory pancreas, as it is merely a part of the pancreas which is separated from the remainder by a depression more marked than usual. The portion of the head lying behind the mesenteric vessels is an example. It forms the lesser pancreas (pancreas parvum).

**Annular Pancreas.**—In rare cases the whole circumference of the duodenum is enclosed in a ring of pancreatic tissue. It may be congenital or a result of invasion of the head of the gland by a growth or from inflammatory changes. It may cause partial obstruction with dilatation of the duodenum above the point of constriction, and usually dilatation of the stomach with hypertrophy of its wall. The patient suffers from symptoms similar to pyloric stenosis.

**Pancreas Accessorium** (*Aberrant Pancreas*).—An accessory pancreas is a mass of pancreatic tissue entirely separated from the pancreas and having a duct of its own. Possibly such isolated masses of gland tissue may vicariously assume the function of the main organ when diseased, and they may cause diverticula or herniæ of the wall of the intestines. Opie[1] reports 10 cases in 1800 autopsies, which he divides into two groups, those lying above the pancreas, in the duodenum and stomach, and those below, in the duodenum and jejunum.

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[1] J. B. Lippincott, Diseases of the Pancreas.

Accessory pancreas has also been found in the ileum in an umbilical fistula, in the mesentery of the duodenum, and in the fat of the great omentum.

The aberrant pancreas averages 1 cm. in diameter, though it has been found as large as 4.5 cm. More than one accessory gland may occur in the same person. About one-third of these glands are situated in the wall of the stomach in the submucosa near the pylorus, and about two-thirds occur in the intestinal wall.

In the latter they are more frequent in the muscular layer, though they are also found in the submucosa. The tissue of the aberrant gland does not differ in histologic features from that of the pancreas.

Among the pathologic changes affecting accessory pancreatic tissue are fat necrosis, chronic interstitial inflammation, and malignant growth.

There are a number of cases recorded in which accessory pancreas is associated with a diverticulum of the intestine occurring in the jejunum, ileum, duodenum, and once even in the stomach.

Such diverticula usually occur in children from less than a year old to those of fourteen years. Intestinal obstruction, acute intraperitoneal inflammation, hernia into the pancreas, both with and without hemorrhagic necrosis, have all resulted from these diverticula.

**Other Anomalies of the Pancreas.**—The pancreas may fall forward, or there may be a congenital displacement of the head of the gland associated with gastroptosis. Wandering spleen may drag the tail of the pancreas into an abnormal position. The pancreas has been pushed down by tight lacing, and has been forced upward by retroperitoneal tumors and aneurysms of adjacent vessels.

A movable pancreas in a man, resulting from a fall, has been reported by Dabrzyki, which gave rise to symptoms resembling those of movable kidney. It has also been met with in hernial sacs in umbilical hernia (both congenital and acquired). Luther reports it in diaphragmatic hernia, and in 1 case it had passed through a rent in the diaphragm into the thoracic cavity.

**Anatomic Variations of the Pancreatic Ducts and Common Bile-ducts.**—Variations in these ducts have an important bearing upon the pathology of the pancreas, and especially on pancreatitis.

*Common Bile-duct.*—The common bile-duct is divided into four portions: the supraduodenal, the retroduodenal, the pancreatic, and the intraparietal or interstitial portion, which last is contained in the wall of the duodenum.

The pancreatic portion, which chiefly concerns us, is from 20 to 25 mm. long, and extends from the lower border of the first part of the duodenum to where it penetrates the second part of this viscus. It is closely applied anteriorly to the posterior surface of the head of the pancreas. It is estimated that in from 62 to 75 per cent. of cases this portion of the common duct is *completely embraced* by the head of the gland and that it runs in a deep groove in the remainder.

It is evident that when the duct passes through the substance of the gland that a swelling, or interstitial inflammation of the head of the pancreas, may compress and even lead to the occlusion of the common bile-duct.

*Variations in the Ampulla of Vater and Terminations of the Ducts.*—Normally, the common bile-duct and duct of Wirsung empty into the ampulla. The pancreatic and common ducts may join a little distance from the duodenum, the ampulla be absent, and the united ducts open into the duodenum by a small flat, oval orifice; or the ducts may open into a concave fossa in the duodenal wall; or the caruncle is well developed, but the ampulla is absent, and the two ducts open side by side. Opie found this latter arrangement in about 11 per cent. of cases. Rarely the common duct unites with the duct of

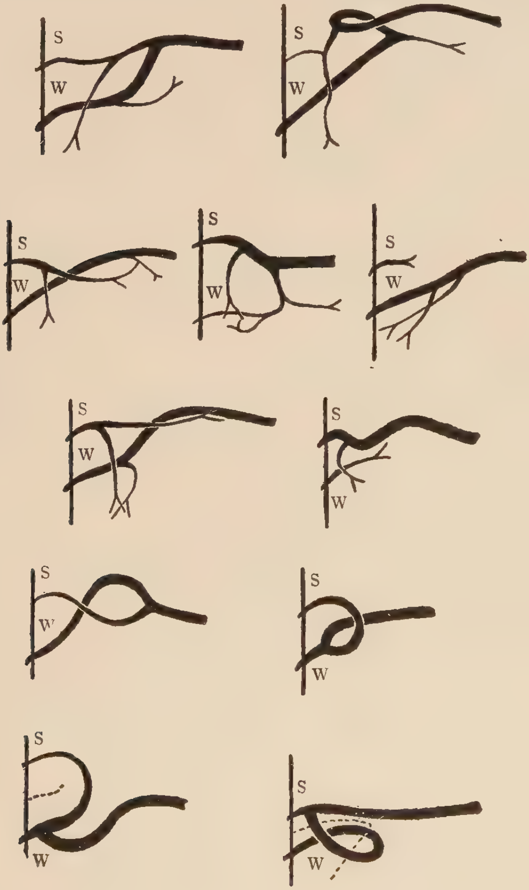

Fig. 356.—Diagram to show the variations in the ducts of Wirsung (W) and Santorini (S). Broken lines, in the plane of the interlobular fissure (after Opie).

Santorini. The bile-duct may open by a circular orifice and the pancreatic duct embrace the bile-duct like a gutter and have an orifice of crescentic shape.

*Variations in the Pancreatic Ducts.*—Opie investigated a series of 100 cases and found both ducts present, though one may have been partially obliterated, or so small that it was demonstrated with difficulty.

In 89 per cent. the duct of Wirsung is the main excretory channel of the pancreas, and in 21 per cent. the duct of Santorini was obliterated near its termination.

Opie found that in 48 cases out of 100 it was possible to inject fluid at low pressure through the lesser papilla into the duct of Santorini. After numerous experiments he concluded that in *more than half* of all cases the lesser duct is *obliterated* at its orifice, or *so constructed* that it cannot assume the function of the larger duct when the latter is occluded.

The varying relations of the duct of Santorini to the duct of Wirsung are shown in Fig. 356.

It must be remembered that the duct of Santorini, even if patent, may be compressed by a gall-stone passing down the pancreatic portion of the common bile-duct.

Three ducts have been found, the additional one being an accessory pancreatic duct opening into the ampulla of Vater.

## SURGICAL RELATIONS

As the head of the pancreas is in intimate relation with the duodenum, disease of the gland may readily invade the small intestine, or the converse may happen. Cancer of the head of the pancreas has obliterated the duodenum to such an extent as to call for a gastro-enterostomy. Malignant growth of the duodenum may invade the pancreas and, finally, give rise to diabetes.

Cysts or tumors of the pancreas may compress the duodenum, or the latter may be involved in a pancreatic abscess which may discharge into the gut.

The stomach lies in front of the pancreas, so that the latter is liable to invasion from ulcer or cancer of the former. Adhesions between the pancreas and stomach, no matter which organ is the source of the trouble, may produce symptoms when the stomach is distended with food, owing to the limitation of the movements of the latter organ, and pain may also result from interference with its peristalsis.

When the pancreas is invaded by or becomes adherent to a malignant growth of the stomach, this increases the danger of operation on the stomach and renders the return of the disease more probable after removal.

In 100 cases of pyloric cancer, Fenwick found the pancreas adherent in 6. With cancer at the cardia, out of 100 cases the pancreas was adherent in 16, and when the cancer was present in the lesser curvature or posterior wall, the pancreas was adherent in 19.

Chronic gastric ulcer, when situated posteriorly, may become adherent to the pancreas and may cause pancreatitis or even produce a pancreatic abscess. The latter may burst into the stomach and discharge therein, producing at the same time an acute gastritis.

On account of the retroperitoneal position of the pancreas in case of suppurative pancreatitis, pus may burrow upward to the diaphragm and downward toward the left iliac fossa. Pus in some cases may be reached from the right or left loin, from the left iliac fossa, or between the ribs, if it presents as a subdiaphragmatic abscess.

The anterior surface of the pancreas projects into the lesser sac of the peritoneum, being covered by its posterior layer. This cavity may be invaded in inflammatory conditions or from an injury to the gland. When filled with fluids it may be mistaken for a true pancreatic cyst. Tumors of the tail of the pancreas and pancreatic cysts in this region are more readily extirpated than when in other regions, as they are less closely connected with the great vessels. The tail of the gland serves as a pedicle. Section and closure of the pancreatic duct results in chronic inflammation of the parenchyma, which is tributary to the occluded duct, and, hence, injury to the pancreas is greatest in proportion to the nearness of the traumatism to the duodenum.

As the pancreas lies at the back of the abdominal cavity, operation upon it is not particularly easy, and various routes are adopted according to indications. One must also remember its relations to the vena cava, the aorta, celiac plexus, spleen, the left suprarenal capsule, the left kidney, portal vein, the common bile-duct, and middle colic artery. The usual method to expose the pancreas is through the gastrocolic ligament, entering the lesser peritoneal cavity between the stomach and transverse colon. If the stomach has sunk downward and the lesion is near the upper border of the pancreas, incision may be made through the gastrohepatic omentum. In some cases one may push up the omentum and transverse colon and reach the pancreas by incision through the lower layer of the mesocolon. One may reach the head of the pancreas for the purpose of pancreatic lithotomy or to examine that part, by incising the peritoneal covering of the duodenum and forcing a way along its side to the head of the pancreas. To reach the posterior surface of the head of the gland in order to expose the pancreatic portion of the common bile-duct one can detach the duodenum from the abdominal wall and lift it inward, separating it from the front of the kidney. Under some conditions extraperitoneal operation may be employed; thus, an abscess of the tail has been opened through the left lumbar region, and a cyst projecting to the right from the head of the pancreas has been opened in the right lumbar region.

## CHAPTER XXXVI

# HISTOLOGY OF THE PANCREAS—PHYSIOLOGY

The pancreas consists of lobules held together by connective tissue. It is composed of branching ducts, terminating in acini that are round or oval (Maziarski).[1]

The acini about the terminal ducts are grouped together to form primary lobules from 1 to 2.5 mm. in diameter. These last are not so regularly arranged or so clearly defined from each other by connective tissue as in animals, and may be indistinctly marked off, and even, in places, fuse with each other.

An island of Langerhans is often situated in the middle of the lobule.

Numerous primary lobules fuse together to form larger secondary lobules, which last are separated by wide bands of loose areolar tissue, in which are contained the larger ducts, the blood-vessels, and the nerves.

They give to the gland its lobulated appearance. They vary in width from 2 to 6 mm.

The arteries and veins lying side by side *do not accompany the ducts*. Numerous secondary lobules grouped together form tertiary lobules. They represent the largest subdivisions on the surface of the gland.

**Connective-tissue Framework.**—The arrangement of the connective-tissue framework of the pancreas is important, in view of the changes which occur as a result of chronic inflammation. The surface of the gland has no capsule, but is covered by a thin coat of connective tissue. Within the substance of the organ the connective tissue is arranged in an interlobular framework which separates the lobes and lobules, and also an intralobular network of fine fibrils which pass between the individual acini and form a plexus. The interlacing fibers form a reticulated basement-membrane which supports the alveolar cells (Fig. 357). Near the islands of Langerhans the processes between the alveoli become thicker and form septa which run into the capsule of the island.

A small amount of elastic tissue is mixed with the fibrous framework, chiefly in the interlobular regions. The ducts throughout are surrounded by a fine network of elastic fibers.

The connective tissue contains some fat and connective-tissue cells. Mast cells are found in the interlobular framework. Cells with polygonal or elongated nuclei lie in the interalveolar connective tissue.

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[1] Anat. Hefte, 1901, Hfte, lviii, 171.

The lobules do not have a definite hilus, but the blood-vessels and ducts enter separately.

**Ducts.**—The duct of Wirsung while passing through the gland gives off lateral branches, most numerous on its upper and lower aspect, which usually enter the main channel obliquely. The branches subdivide and give off lateral twigs, which ultimately penetrate the tertiary lobules.

The pancreatic ducts and their large branches have a wall of connective tissue containing both white and elastic fibers, and lined by columnar cells. Some believe these cells discharge a secretion which mixes with that of the alveoli. In the wall of the duct of Wirsung there are structures having the character of mucous glands. The medium-sized ducts within the secondary lobules have little connective tissue in their walls, and are lined by cubic or low cylindric cells; the smallest ducts, about which the acini are grouped, are formed by a layer of flat epithelium with a large oval nucleus. They appear spindle shaped on longitudinal section.

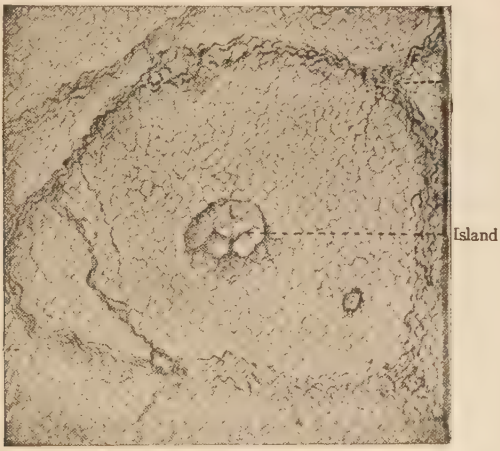

Fig. 357.—Piece digestion of a human pancreas, showing the limiting membrane of a lobule and the reticulated basement-membranes of the alveoli. In the center is an island of Langerhans with its capsule of trabeculæ (× 26) (after Flint).

**Acini.**—The shape of the acini varies in different animals. In the human pancreas they are round or oval, and at times have a lobed surface. They do not branch.

The cells forming an acinus are shaped like a truncated pyramid with the apex toward the lumen of the acinus (Fig. 358). They constitute the secreting cells.

The apical zone contains refractive zymogen granules. There is a basal zone, which is homogeneous except for striations parallel with the long axis of the cell.

The granules of the apical zone stain with certain acid dyes, while the protoplasm of the base stains deeply with hematoxylin. There is a nucleus situated near the base of the cell. It is spheric or, occasionally, oval, and has a well-defined nuclear membrane. There is usually a large nucleolus.

The centro-acinar cells lie in the acini in contact with the apices of the secreting cells. They are fusiform, occasionally flat, and may have short projections penetrating between the secreting cells. The protoplasm is nearly homogeneous and the nucleus is small, oval, and rich in chromatin. These cells resemble those of the terminal ducts, with which they seem to be continuous. There are at times scattered fat drops in the acini.

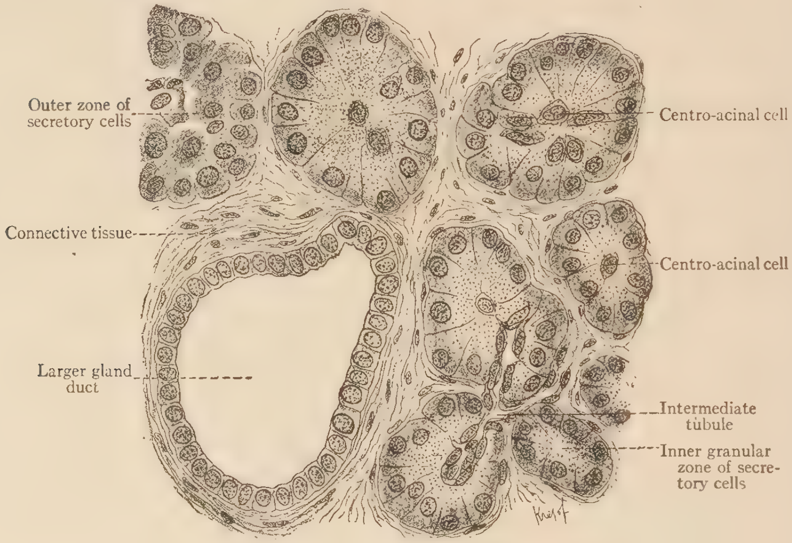

Fig. 358.—From section through human pancreas; × 450 (sublimate) (Böhm and Davidoff).

**Changes with Secretion.**—Haidenhain[1] has demonstrated in dogs that changes occur in the cells of the acini during the period of active secretion of the pancreas. From six to ten hours after a full meal the inner zone, which contains the granules, decreases in size until it occupies only the tip of the cell. The outer zone increases in height, but not as rapidly in proportion as the inner zone decreases, hence, the cell becomes smaller.

From the tenth to the twentieth hour after food the cell increases in volume, the apical zone increases in size through accumulation of granules, and the basal zone diminishes in size.

In the active or "discharged" gland the granules are comparatively scanty, while in the resting stage they are abundant, and the precursor of trypsin is abundant in the gland of a fasting animal.

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[1] Arch. f. d. ges. Physiol., 1875, x, 557.

**Islands of Langerhans.**—These structures (Fig. 359), also known as the interacinar islands, are usually round or oval, and their outlines are frequently defined by a circle of thin connective tissue.

In others the shape is less clearly defined. Their size may be from 75 to 175 microns in diameter and occasionally as large as 0.5 mm. DeWitt[1] estimates that their tissue constitutes from $\frac{1}{125}$ to $\frac{1}{25}$ of the total volume of the pancreas.

Opie[2] finds that the islands of Langerhans are more numerous in the splenic end or tail; in all, slightly less than three and a half times more numerous in these regions than in other parts of the pancreas.

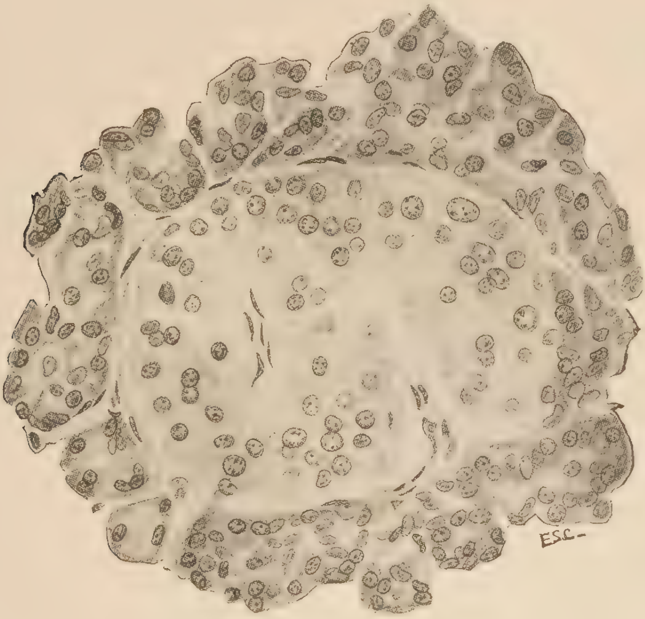

Fig. 359.—Island of Langerhans of the human pancreas (Opie, "Diseases of the Pancreas").

*Position of the Islands.*—This is not constant, though they are most frequently situated in the center of a lobule.

*Cells of the Island of Langerhans.*—The cells are polygonal, and are smaller than those of the acini. They have a large round or an oval nucleus, with several nucleoli. The cell body is usually well defined. They are unstained by nuclear dyes, unlike the secreting cells of the acini, while with eosin their protoplasm stains a bright pink. The nuclei vary in size, and occasionally very large nuclei are found, larger than the adjacent cells.

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[1] Jour. of Exper. Med., 1906, viii, 193.

[2] Johns Hopkins Bulletin, 1900, xi, 205.

The cells lie in columns, between which there are anastomosing capillaries, though at times they are packed together, and the nuclei lie side by side.

Fat droplets are found in the islands of Langerhans.

The protoplasm of the cells of the island is not entirely homogeneous, but contains small granules, staining with eosin, safranin, gentian, violet, and fuchsin.

Lane[1] has demonstrated two varieties of cells, one soluble and the other fixed by Müller's fluid with bichlorid.

*Blood-vessels.*—The capillaries of the island form a glomerulus of tortuous vessels (Fig. 360) which freely anastomose.

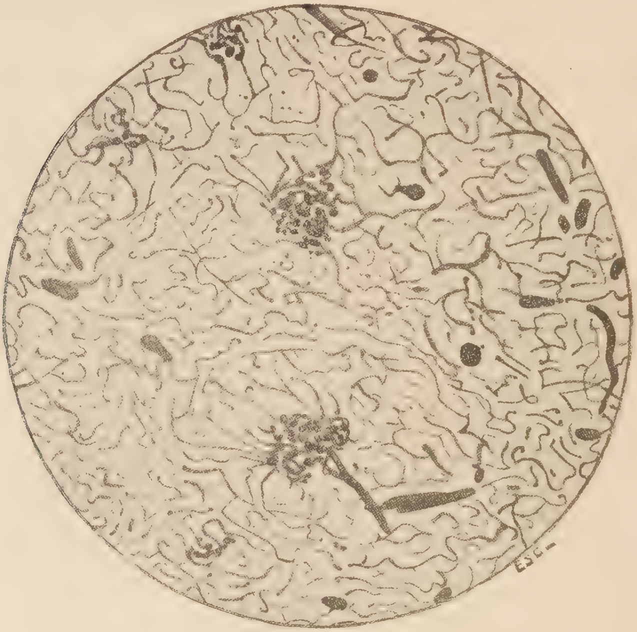

Fig. 360.—Blood-vessels of the pancreas injected in order to show the glomerular arrangement of capillaries in the islands of Langerhans (Opie, "Diseases of the Pancreas").

A single afferent vessel does not enter the glomerulus, as it does in the kidney. There are numerous anastomoses, so that the network within the island is continuous with the capillaries between the acini.

There is no evidence in the human subject that there is *any connection* between the islands and the *duct system of the gland.*

Opie holds that there is no proof that any transition occurs from acini into islands of Langerhans.

*Intralobular Framework.*—The connective tissue between the acini near the islands of Langerhans becomes thicker, and forms septa which run into the capsule of the island.

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[1] Amer. Jour. of Anat., 1907, vii, 409.

The capsule is composed of many small fibers, which are connected on the one side with the alveolar network, and on the other with septa, which stretch across the space within the island, subdividing it into small lacunæ, and which act as a support for its cells.

In the human subject the age and the condition of the body seem to be the chief factors in determining the amount of the intra-insular connective tissue.

DeWitt shows that in the pancreas of the newborn infant there is no connective tissue in the islands; in a four-year-old child there is a delicate capsule and sheaths surrounding the blood-vessels; while in the adult the condition was found as described. The increase accompanying advancing age suggests the condition of fibrosis, which occurs in other organs under similar conditions.

*Function of the Islands of Langerhans.*—On account of the intimate relation of the columns of the epithelial cells of the islands to the capillary network, it is believed the islands furnish some substance to the blood, known as the "internal secretion of the pancreas."

A study of the pathologic changes associated with diabetes mellitus has shown that the islands of Langerhans control the assimilation of sugar.

## PHYSIOLOGY OF THE PANCREAS

**The Pancreatic Juice.**—The pancreatic juice is a clear, watery, alkaline fluid, without odor, with a specific gravity of 1.0075 to 1.0098. It consists of approximately 98.5 water and 1.5 solids. One-third to two-thirds of the solids are ash. It contains a small amount of coagulable protein and enzymes, which play an important part in the digestion of protein, carbohydrates, and fat.

The gland is inactive when fasting. Secretion begins directly after a meal and reaches its maximum within three hours.

The quantity and character of the pancreatic juice depends on the character of the food. Meat causes a rapid secretion of a juice poor in solids, while bread, a slower secretion, but in greater quantity. Milk produces the flow of a small amount of concentrated juice with a low alkalinity.

**Cause of Pancreatic Secretion.**—Bayliss[1] and Starling, isolating a loop of the jejunum and severing all its nerve connections, the blood-vessels only remaining, demonstrated that the introduction of hydrochloric acid into the loop produced an abundant flow of pancreatic juice. Evidently some chemic substance which was transported by the blood from the loop to the pancreas caused the secretion Acid introduced into the circulation did not excite secretion, and hence it was evident that acid introduced into the intestines caused the mucosa of that viscus to elaborate some substance which was absorbed by the blood, was carried to the pancreas, and thus excited its activity.

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[1] Jour. of Physiol., 1902, xxviii, 325; 1903, xxiv, 174.

Moreover, the epithelial cells of the intestinal mucosa treated with the acid yielded an extract which, when it was introduced into the circulation, caused an active flow of the pancreatic juice. They named this substance "secretin." As an extract of the cells alone, when injected into the circulation, did not produce pancreatic secretion, it was evident that the acid activated some substance in the cells. They called this "prosecretin."

The acid evidently converted the prosecretin into secretin, which last was carried by the circulation and excited the pancreas into activity.

Pawlow believed that nervous mechanism, stimulation of the vagus, has a direct influence on pancreatic secretion. Other theories have also been advanced, but the work of Bayliss and Starling is considered conclusive. *Fats and soaps also stimulate pancreatic secretion,* but their action is less marked.

The epithelial cells of the duodenum and of the jejunum chiefly contain prosecretin. Wertheimer[1] and Lepage found that the acid caused the greatest flow of pancreatic secretion when it was introduced into the duodenum; a less intense flow, lower in the small intestine, and that it disappeared when the lower part of the ileum was reached.

**The Ferments.**—The three chief ferments of the pancreas are trypsin, amylopsin, and steapsin. Some claim there is a lab ferment, on account of the ability of the pancreatic juice to coagulate casein, while others impute this action to the trypsin. Other observers believe there is a "lactase ferment," because of its supposed power of splitting milk-sugar into galactose and dextrose.

*Nuclease.*—Nucleic acid is, for the great part, precipitated in the stomach and dissolved in the intestines. This solution of the nucleic acid, which is caused by the pancreatic juice, is attributed to a pancreatic enzyme, "nuclease."[2] Others believe it to be simply a property of trypsin.

*Trypsin.*—Trypsin does not exist as such in the secreting glands of the pancreas, and extracts of the fresh gland do not act upon protein. It is believed that the pancreas contains trypsinogen, since the juice which is obtained from the pancreatic duct after the use of secretin contains this enzyme. The intestinal juice (succus entericus) contains an enzyme, "enterokinase," which transforms the inactive trypsinogen into active trypsin. Pawlow, on the other hand, claims that the pancreatic juice of a dog fed on a meat diet contains trypsin.

Trypsin is a proteolytic ferment, and not only forms albumoses and peptones from protein, but the action is more prolonged, so that they are transformed into amino-acids. The action is most powerful in a weakly alkaline solution, but it will act in a neutral or even faint acid medium. Free mineral acids destroy the ferment. Organic acids are less harmful.

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[1] Jour. de Physical et de Patholgén, 1901, iii, 335.

[2] Umber, Zeit. f. klin. Med., 1901, xliii, 282.

Collagen, the constituent of connective tissue, is not acted on by pancreatic juice unless it has been previously boiled with water or has been acted on by dilute acid, hence, connective tissue is not digested if the stomach has been removed or if the secretion of the acid of that organ is interfered with.

Gelatin is converted into gelatin peptones and elastin is dissolved. Mucin and nucleoproteins, after preliminary cleavage, undergo digestive changes. Starling has shown in the fasting animal that a mixture of the pancreatic juice with the intestinal secretions may give rise to inflammation and erosions of the intestinal wall. In the case of pancreatic fistula similar effects on the skin are produced.

*Steapsin.*—The digestion of fat is peculiarly a function of the pancreas. Neutral fats, we must remember, owing to a gastric lipase ferment, undergo slow changes in the stomach, and fine emulsions, as occurring in the yolk of eggs and milk, are more completely digested. This is of importance in the case of infants before the lipolytic function of the pancreas is developed, and also in adults whose pancreas is diseased.

Steapsin, the fat-splitting enzyme of the pancreas, acts in an alkaline, neutral, or acid reaction. It splits neutral fats into fatty acids and glycerin. When an alkali is present, the fatty acids form soluble alkaline soaps. These, together with the glycerin, are absorbed by the epithelial cells of the walls of the intestines, within which they are synthesized to form neutral fat.

Steapsin can also decompose a number of esters of fatty acids, such as ethyl-butyrate, and the latter can be used as a test for the presence of the fat-splitting enzyme. The amount of acid formed measures its activity. The action of the enzyme is reversible.[1]

Bile increases the activity of the fat-splitting enzyme several times. Its method of action is unknown.

*Amylopsin.*—This is an amylolytic enzyme which converts starch into dextrin, and, finally, into maltose. Little, if any, dextrose is formed from starch, and invertin is not produced. Amylopsin acts in the presence of a weak acid and is somewhat inhibited by a weak alkali. Amylopsin is not present in the pancreas of the newborn infant, and does not appear until more than a month after birth.

**Autodigestion of the Pancreas.**—Self-digestion of the pancreas after death of the subject occurs in about 50 per cent. of cases. It is attributed to the proteolytic enzyme.

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[1] Kastle and Loevenhart, Amer. Chem. Jour., 1901, xxiv, 491.

## CHAPTER XXXVII

# METHODS OF DIAGNOSIS IN PANCREATIC DISEASE

### TESTING THE FUNCTIONS OF THE PANCREAS

THE pancreas has two secretions, an internal and an external. The former passes into the blood and is concerned with the metabolism of sugar. It also appears to have an antagonistic influence on the secretion of the adrenals, as Loewi has shown by experiment that, in dogs whose pancreas had been removed, dilatation of the pupil took place after instillation of adrenalin into the eye. The external secretion, which contains the proteolytic, fat-splitting, and amylolitic ferments (and, as some believe, a lab ferment and nuclease), will first be referred to in this chapter.

In order to properly test the digestive capacity of the pancreatic juice, and to arrive at some definite conclusion as to whether this organ is suffering from a *temporary disturbance* of one or more of its functions, or whether there is *permanent disability, i. e.*, a diseased condition, the author believes that numerous factors must be taken into consideration, and that the diagnosis must often be made by the process of exclusion.

**Temporary Disturbances of Function.**—In *acute febrile conditions*, in typhoid fever, for example, there is a disturbance of all the digestive secretions, salivary, stomach, liver, intestine, and pancreas. As a result, the digestive function of the pancreas is diminished. This has been previously referred to by the author under Typhoid Fever, and is, from his viewpoint, a scientific reason for the avoidance of excessive forced feeding, 4500 to 5500 calories, during the active stage of this disease. Duodenitis may complicate the typhoid. There may be temporary disturbances of the pancreas for any one or for all of the functions, to which reference is made under Intestinal Dyspepsia.

Catarrh of the duodenum may *temporarily* disturb the functions of the pancreas by interference with the formation of its activating prosecretin, or its conversion into secretin, and it also interferes with elimination of the enterokinase which activates the trypsinogen. The catarrh may spread into the common bile-duct and into the pancreatic duct; in the latter instance interfering with the pancreatic secretion, and also partially or completely block the common duct. Obstruction of the pancreatic duct, from the pressure of tumors or from calculi in the common duct, or similar conditions affecting the pancreatic ducts, are the most frequent causes which may give rise to a more or less complete absence of the pancreatic secretion from the intestines. Atrophy, fatty degeneration, or sclerosis of the pancreas may result. With interference with the biliary excretion, the digestion of fat would be markedly diminished and the pancreas might be involved.

A negative sublimate test would suggest that suppression of bile was the cause. This further shows the necessity of care in diagnosis. Anemia, nervous exhaustion, fright, grief, mental overwork, worry or fatigue from excessive muscular work, or sexual excess, Herter gives as further causes of temporary disturbance of the pancreatic secretion.

With chronic duodenitis, which extends into the ducts, permanent damage to the pancreas may result.

*Other Conditions Obscuring the Diagnosis.*—With tuberculosis of the intestines and peritoneum, sprue, and amyloid disease, fat absorption is impaired, and these facts must be held in consideration. Enterocolitis with diarrhea, the use of purgatives, and excesses in diet in reference to meat, fats, and carbohydrates, must also be considered. The gastric secretion *under certain conditions influences fat digestion* when the fat is *contained in meat.* For example, collagen, the chief constituent of connective tissue, is not acted on by the pancreatic juice unless previously boiled with water or unless it has been acted on by an acid. Deficiency or absence of hydrochloric acid in the stomach, therefore, may cause the appearance of an abnormal quantity of fat in the feces. On the other hand, fats and soaps stimulate pancreatic secretion to a certain degree, and this undoubtedly accounts for the fact that in achylia gastrica, with the absence of hydrochloric acid, nutrition may be improved by the administration of fats, such as cream, butter, milk, etc.

Therefore, in testing the digestive capacity of the pancreatic juice, these facts must be taken into careful consideration. *At the end of the chapter the author recommends the methods which he considers most practical.*

There are two methods of **obtaining the pancreatic ferments,** the direct and the indirect.

**Direct Methods.**—Boas has obtained the duodenal contents by passing the stomach-tube into the empty stomach and massaging near the duodenal region, thus forcing the juice into the stomach; while Hemmeter and Kuhn have passed the stomach-tube directly into the duodenum. These procedures are uncertain.

Einhorn has employed the duodenal bucket and has also endeavored to catheterize the duodenum. They are impractical.

There are two methods more recently introduced by which it is possible to secure in *some cases,* but *not in all,* the duodenal contents. There are two instruments reported almost synchronously, the duodenal tube of M. Gross[1] and the duodenal pump of Einhorn.[2]

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[1] N. Y. Med. Jour., Jan. 8, 1910.

[2] Med. Rec., Jan. 15, 1910.

**The Gross Tube.**—This instrument is depicted in Fig. 361.

It consists of a perforated round metal ball about twice the size of a pea, to which is attached a thin rubber tube 0.2 cm. in dimaeter and 125 cm. in length, marked every 10 cm. To this is attached a glass receptacle (bulb), which is connected by a length of tubing with a mouth-piece, which the operator can use to aspirate, or to which an aspirating bulb can be attached.

Gross recommends the following method:[1]

"*Test-meal.*—The patient is given in the morning a tumbler (250 c.c.) of milk and water, equal parts. This mixture does not cause a great flow of hydrochloric acid and contains sufficient fat to stimulate the pancreatic secretion. Half an hour later introduce the duodenal tube.

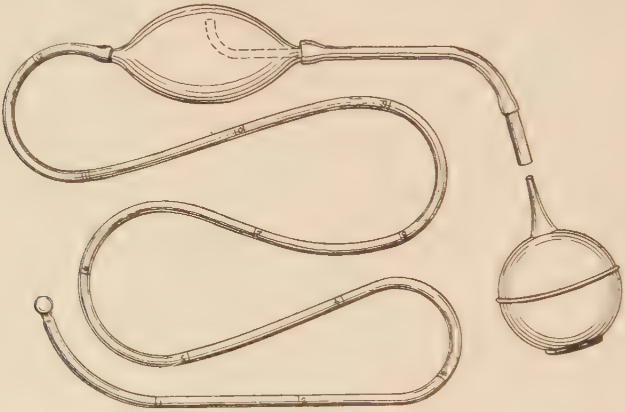

Fig. 361.—Gross duodenal tube.

"*Technic.*—The patient swallows the ball and tube, previously wet in water, until the mark 45 cm. reaches the lips. Then blow lightly through the tube into the stomach, so as to smooth the tube out and that it will hang freely in the cavity. The patient should then lie down and slowly turn over on the right side, in which position, after a few minutes, the tube is allowed to glide down through the mouth without swallowing, following the pull of the ball, until the mark 60 cm. has been reached. After five or ten minutes the first aspiration may be made, which usually shows contents of a slightly yellow tint. The patient, with the mouth still partly open, should not make any swallowing movements. Gradually the tube will be drawn down to the 65 or 70 cm. mark.

"A second aspiration is then made, and usually a yellowish fluid, free from casein, will be aspirated, giving a weakly acid or acid reaction. One should wait a little longer and, after several aspirations, the yellow aqueous contents of the duodenum will usually be secured, giving a neutral or alkaline reaction, and at times a greenish-yellow fluorescence."

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[1] Jour. Amer. Med. Assoc., April 23, 1910.

*Precautions.*—Occasionally the aspirated fluid remains acid, probably due to hyperchlorhydria, and, therefore, neutralization of duodenal contents may occur lower down. As a check, give a cup of coffee with the tube in place. Aspiration should be performed, and if the fluid is still green, the contents are duodenal. This may be proved by withdrawing the tube a short distance and reaspirating, when the coffee will appear. Gross holds that unless there is a mechanical obstacle at the pylorus, it is *usually* possible to pass through the pylorus with the tube and into the duodenum within an hour.[1]

**Einhorn's Duodenal Pump.**—This instrument, similar in principle to the one already described, is readily understood from the illustration (Fig. 362).

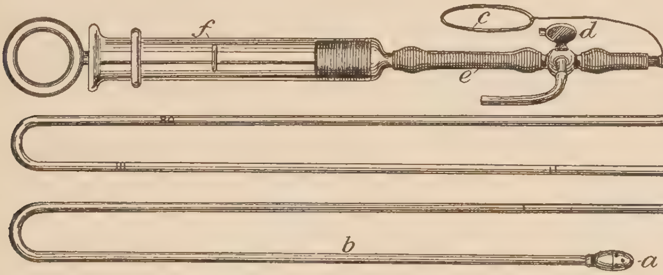

Fig. 362.—Einhorn's duodenal pump: *a*, Metal capsule, lower half provided with numerous holes, the upper half communicating with tube *b;* I, II, III, marks of I = 40, II = 56, III = 70 cm. from capsule; *c*, rubber band with silk attached to end of tubing, which can be placed over the ear of the patient; *d*, three-way stop-cock; *e*, collapsible connecting tube; *f*, aspirating syringe.

It has three markings—40 cm. (cardia), 56 cm. (the pylorus), and 70 and 80 cm. distance from the capsule. The duodenal contents are aspirated out by the syringe, the cock turned, and they are then ejected into a vessel, and so on.

The patient in the fasting condition drinks a cup of tea with sugar and without milk, and then about one-half to three-quarters of an hour later the capsule and tube are lubricated in water and gradually swallowed, the effort being aided by drinking about half a glass of water. To be sure that the capsule has entered the stomach and is not kinked, one can aspirate and determine the presence of gastric contents. A syringeful of water is then forced through the tube and then one of air, and the tube is then shut off by the stop-cock and the thread hitched over one ear. The patient should not close the lips or teeth and should read for about an hour.

When the tube reaches the mark 70 cm. at the lips, aspiration is attempted. If the capsule is in the duodenum, on aspirating one generally obtains a golden-yellow watery liquid of alkaline reaction, somewhat viscid in consistency.

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[1] N. Y. Med. Jour., July 9, 1910.

Einhorn[1] now recommends, as soon as the flow commences, that the piston of the syringe be removed and the barrel be kept low, so as to siphon out the fluid.

At times one does not obtain the duodenal contents, as the tube may coil in the stomach. If acid gastric contents are obtained, withdraw the tube to the 56 cm. mark, wash it out with water, and then blow in air, and in half an hour try again. When the tube lies in the stomach, it does not collapse on aspiration; when it lies in the duodenum, it collapses and aspiration is slower.

As a further test, one can give the patient a little milk with the tube in place, and if the capsule lies in the duodenum no milk will appear.

The writer finds these methods of obtaining the duodenal contents are by no means always successful.

Patients who have an irritable pharynx and esophagus will not retain the instrument for the time necessary to secure the duodenal contents. In cases where there is pyloric stenosis, even of no marked degree, in atonic dilatation of the stomach, and often in atony of the stomach, the procedure may prove a failure. With normal conditions of the stomach it may sometimes be unsuccessful, or only a drop or two or no contents be extracted, even when the tube is apparently in the duodenum.

Moreover, the pancreatic ferments are active, even in very small quantities, and as long as there is *any pancreatic tissue functionating* and possibility of its escape into the duodenum, one would expect to *secure the reactions* due to their presence.

This last view is referred to by Deaver.[2]

The *Schmidt-Strassburger diet* might show *marked deficiency in pancreatic function* for *an average diet*, while *the direct test indicated the pancreatic ferments to be present.*

The writer believes the *Tests of the Intestinal Functions should be used in every case* (p. 492).

## EXAMINATION OF THE DUODENAL CONTENTS

**Bile-pigment.**—Add 1 c.c. of fuming nitric acid to 1 c.c. of the duodenal contents in a test-tube and note if Gmelin's reaction occurs. With sufficient bile, the juice turns green.

The following simple tests are suggested by Einhorn for the ferments:[3]

**Steapsin.**—Take 1 drop of neutral milk, 2 drops of water, and 2 to 3 drops of the duodenal contents, which should be neutralized if the reaction is acid, and add a small piece of blue litmus-agar. Place this in a small test-tube and keep it at blood temperature. If steapsin is present the agar piece turns red in twenty to thirty minutes, owing to the development of fatty acids. The writer finds an excellent *incubator is a Thermos bottle* containing water at 99° to 100° F., in which *the corked test-tube can be placed.*

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[1] Jour. Amer. Med. Assoc., July 2, 1910, vol. lv, pp. 6–8.

[2] Ibid., April 15, 1911.

[3] Med. Rec., Jan. 15, 1910.

**Trypsin.**—If the fluid (duodenal) is acid, neutralize it and place in it a small piece of the white of a hard-boiled egg. Keep it a few hours at blood temperature. If trypsin is present, the albumin disappears.

**Amylopsin.**—To test for diastase, use a boiled starch solution or starch-paper. Either mix the duodenal contents with equal parts of starch solution or insert in it a strip of starch test-paper, and leave it at blood temperature. In half an hour to an hour add a weak iodin solution, and if dextrin is present, the starch solution or paper turns blue; if erythrodextrin is present, we have a red color.

One must always note with litmus-paper the reaction of the duodenal contents. It is usually alkaline, though occasionally HCl is present, giving an acid reaction.

Gross[1] recommends the following, though more complicated tests are suggested:

**Amylopsin.**—A quantity of a 1 per cent. aqueous solution of Kahlbaum's soluble starch is heated in the incubator to 55° C.; also a number of empty test-tubes are heated up; 5 c.c. of the heated starch is put in a hot tube, 4 drops of duodenal juice are added, and the tube shaken for about a minute; $\frac{1}{2}$ c.c. of a 250th normal iodine solution is then added.

This normal iodin solution consists of an aqueous solution of equal parts of iodin, iodid of soda, potassium iodid, and ammonium iodid, 1 : 250.

If no amylopsin is present, the solution becomes blue; or green if bile is present; if traces of amylopsin are present, it turns blue violet; if marked traces, then red violet; if the quantity is normal, then pale pink; and if an excess, then it is colorless.

If to this solution Fehling's alkaline solution is added, it will become colorless. On the addition of Fehling's copper solution on boiling, the sugar reaction occurs, if such is present.

**Steapsin.**—Melt some fresh butter and abstract the clean, pure fat mixture with a little 0.1 per cent. aqueous carbonate of potash and phenolphthalein solution, and then shake and titrate with liquor sodæ until a distinct red coloration remains. This solution is then heated in the incubator to 55° C. Then 5 c.c. of this is shaken with 5 drops of intestinal juice in a heated test-tube.

If there are normal quantities of steapsin, the red color will disappear in from two to five minutes. The quantity of active steapsin can be estimated from the rapidity of this discoloration.

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[1] Med. Rec., May 21, 1910; ibid., Nov. 12, 1910.

**Trypsin.**—Mix a solution of 0.07 per cent. copper sulphate and 0.1 per cent. sodium carbonate with a few drops of Fehling's alkaline solution and a 5 per thousand of casein. Heat this in an incubator to 55° C. Shake up 5 c.c. of this with 5 drops of the duodenal contents. The color is at first blue or green if bile is present. Depending on the quantity of trypsin, it will become more or less red violet, and after a few minutes, pink.

**Indirect Methods of Obtaining the Ferments.**—Pawlow[1] discovered that if he administered oil to dogs having a gastric fistula, within one or two hours he was able to obtain an emulsion containing bile and pancreatic juice due to the regurgitation of the duodenal contents into the stomach.

Boldyreff[2] used a combination of fatty acid in oil and secured a regurgitated fluid containing trypsin.

Levinsky[3] advises giving half a teaspoon of magnesia usta twenty minutes before the oil, and a second dose twenty minutes after, to neutralize the acidity of the stomach-contents. This *is the method that is usually employed.*

The technic is as follows:

First determine the acidity of the gastric juice and if free hydrochloric acid is present; then on an empty stomach one-half teaspoon of magnesia usta is given in water, and this is followed in twenty minutes to half an hour by 200 c.c. of olive oil, and twenty minutes later by another half teaspoon of magnesia usta. Three-quarters of an hour later the stomach should be aspirated, and the fluid, which is often greenish yellow, should be placed in a separating funnel. The oil rises to the surface. The lower part of the fluid is tested for trypsin and the other ferments.

Abderhalden and Schittenhelm,[4] in order to determine the presence of trypsin, advise the use of the polypeptid glycyltyrosin. This optically active peptid cannot be split by pepsin, but is acted on by trypsin, the tyrosin crystallizing out. They have also employed a solution of silk peptone, which contains 40 per cent. tyrosin, and observe the rotation of light by the polariscope.

The author believes the simpler methods to determine the presence of the ferments to be preferable for the general practitioner.

**Securing the Ferments from the Feces.**—The O. Gross[5] method is usually employed. He recommends a high protein diet for two to three days previous to the test, and a mild saline purgative the morning of the day when the test is to be made, so as to hurry the stool through the intestinal tract, that there may be less loss of ferment by bacterial action. The stool should be examined as soon as possible.

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[1] Arbeit der Verdauungsdrüsen, Wiesbaden, 1898, pp. 159–161.

[2] Report of International Physiolog. Congress, Brussels, 1904.

[3] Deutsche Medezin Wochenschr., 1907, p. 403.

[4] Zeitschr. für Physiolog. Chemie., lxi, p. 421, 1909.

[5] Deut. Med. Woch., 1909, Nr. 16.

A solution of ½ gram of casein (Grubler) is dissolved in 1 liter of sodium carbonate solution (1 : 1000). A portion of the stool is rubbed up with three times the amount of soda solution and filtered; 10 c.c. of this filtrate are placed in a small flask with 100 c.c. of the casein soda solution, and a little chloroform is added. The mixture is then put in the incubator at 38° C., and after four and eight hours respectively is tested by taking out 10 c.c. and adding to it with care, drop by drop, a 1 per cent. acetic acid solution. If the solution is clear, the casein has been digested; if it becomes cloudy, the flask is put back into the incubator and tested again after twelve, fourteen, and twenty-four hours. At the end of this time, if no digestion has taken place (if the solution is still cloudy), trypsin is absent or very markedly diminished.

Frank recommends the use of the Berkfeldt filter under pressure before this test, in order to insure a clear filtrate, but it is difficult to clean, and some of the ferment is liable to be lost.

When we estimate the value of the casein method we must remember that the *erepsin in the intestinal juice* and bacterial action have some digestive effect on this product. Attempts have been made by Schittenhelm[1] and Frank to isolate erepsin.

**Müller's Serum Plate Method for Trypsin.**—This method[2] is as follows: A glycerin injection is first given to empty the lower bowel. The patient is then given a test-meal of 150 gm. of meat and 150 gm. of potato gruel, and several hours later a strong laxative is given. If the stool which results is thick, rub it up with glycerin water (10 in 1000). If it is acid or neutral, render it alkaline with soda. Small drops of the feces are then placed on the surface of a serum plate. This last consists of a Petri dish covered with a fairly thick layer of coagulated blood-serum. This is then placed in an incubator at 50° to 60° C.

If an active proteolytic ferment is present, small depressions soon appear in the serum due to digestion by the enzyme. In the absence of the ferment no depressions occur.

The ferment contained in leukocytes will produce pitting of the plate, so that if pus or blood are present in the stool the specimen should be discarded.

If large quantities of fat are present, it may interfere with the test. The fat should be first removed by shaking out with ether.

**Schmidt's Nuclei Test.**—Adolf Schmidt[3] introduced a test which depends on the power of the pancreatic juice to dissolve the nuclei contained in meat. Small pieces of meat are hardened in absolute alcohol and enclosed in small gauze bags. These are soaked in water for some hours and then swallowed. The stool is searched for the bags, and the contents are frozen, cut, and then stained with hematoxin, and examined for the presence of nuclei. If they are not found, one presumes the pancreatic juice has digested them. If they are present, it is assumed that the juice is absent. There is a question as to how much the digestion of the nuclei is affected by *erepsin*, and Klieneberger[1] states that nuclei have been found intact when the pancreas was not involved. Einhorn disputes the test. Moreover, in cases of diarrhea the bags may pass through so quickly that the pancreatic juice has no chance to act, and in cases of marked intestinal putrefaction the nuclei may be destroyed.

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[1] Zentralblatt der Gesamt. Physiol. und Pathol. des Stoffwechsels, 1909, Nr. 23.

[2] Medezin Klinik, 1909, Nr. 16, p. 573.

[3] Deut. Med. Woch., 1899, Nr. 49, p. 811.

**Sahli's Test.**[2]—He employs a gelatin capsule hardened with formol and containing 0.5 gm. of iodoform, or one with 0.25 gm. of salol. The capsule is given with the Ewald test-meal, and normally iodin can be recovered in the saliva, or salicyluric acid (from the salol) in the urine in four to six hours.

A strip of starch-paper moistened with the saliva and touched with fuming nitric acid gives a violet or blue color for iodin—from the iodoform capsule. The urine tested with neutral ferric chlorid solution gives a violet color when the salol test is used. Sahli prefers the salol test. Two or three of the salol capsules are given. One should be sure of the condition of the motor function of the stomach, and this should be first investigated. Absorption from the intestines would be interfered with by a catarrhal condition, and in case of diarrhea the capsule may pass through unchanged. All these conditions must be considered.

**Sodium Fluorid Fibrin Test.**—This method, devised by Huber and Arthur,[3] consists in soaking strips of fibrin in a 2 per cent. solution of sodium fluorid for twenty-four hours at 40° C. The material to be tested is then diluted with an equal amount of the sodium fluorid solution, and the whole is digested for a considerable time at 40° C. If trypsin is present, crystals of tyrosin appear.

## THE FATS IN THE FECES

Normally, little more than 5 to 10 per cent. of fat escapes absorption. Impaired assimilation of fats and proteins is more readily recognized than alterations affecting the digestion of the carbohydrates.

Experiments on animals have demonstrated that extirpation—complete or partial—or destruction of the pancreas is attended by a defective absorption of fat, except of the emulsified fat of milk, in which case a greater proportion was assimilated. Similar disturbances of digestion occur in human cases, where the pancreas has been partially destroyed by chronic inflammation, by cysts, and by new growths.

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[1] Medizenische Klinik, 1910, p. 89.

[2] Lehrbuch der Klinischen Untersuchungs Methoden, 1909, p. 570.

[3] Arch. de Physiologie, 1898, p. 622.

Occlusion of the duct caused by calculi or by tumor, followed by degenerative changes in the gland, may produce the same effect.

Opie[1] shows that *even when the duct of Wirsung is obstructed the pancreatic juice may reach the intestine through the duct of Santorini* in about three-fourths of all individuals, and that often *digestion proceeds with no manifest impairment.*

In estimating whether a true fatty stool (steatorrhea) is present, one must exclude the acholic stool due to the absence of bile-pigment. The acholic stool is grayish white, ash gray, or of clay color, due to the *absence of bile-pigment*. Dilute fecal matter added to a strong solution of bichlorid of mercury is colored red when unchanged bile-pigment is present. *A negative sublimate test suggests the suppression of bile through occlusion of the bile-duct.* There is considerable fat in the acholic stool, seen microscopically as needle-shaped crystals or in sheaves of crystals, or, at times, in fat-droplets. Owing to the absence of *bile from the intestinal tract*, the emulsifying power of the pancreatic juice is diminished and excess of fat appears.

Moreover, an abnormal quantity of fat may appear in the stools of healthy individuals after the ingestion of great quantities of fat. The stool may also be devoid of color or grayish white where there is no jaundice and no occlusion of the bile-ducts, with stercobilin present and large quantities of fat, in conditions where the absorption of fat is impaired, such as with tuberculosis of the intestines and tuberculosis of the mesenteric glands. Fat may also appear in excess in the stools in extensive catarrh of the intestines and in conditions accompanied by active peristalsis. Only in the *absence of the above conditions is the presence* of excessive fat in the stools an aid to the diagnosis of pancreatic disease.

**Steatorrhea.**—This term is strictly applied to those cases in which the fat is discharged in the feces as an oily yellow fluid or in isolated masses, visible to the naked eye.

Opie states there are some cases of pancreatic disease in which the feces are clay colored or grayish white, frequently with a metallic or asbestos-like appearance, and that the increased quantity of fat can be demonstrated only by microscopic or chemic examination. These stools contain fatty acids, neutral fats, and soaps.

Steatorrhea may follow the loss of pancreatic secretion, and Fitz demonstrates its diagnostic value when it is present. Up to 1903 he collected only 29 cases in which there were fatty stools in patients in whom there was conclusive evidence of pancreatic disease.

Theodor Brugsch[2] states that in pancreatic disease, without jaundice, the average loss of fat is 64 per cent.; with mild jaundice associated, the loss is 72 per cent. While in cases in which the bile is completely shut off, the loss of fat is 87 per cent.

A case of carcinoma has been reported by Robson in which

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[1] Disease of the Pancreas.

[2] Lehrbuch Klinischer Untersuchungs Methoden, 1909, p. 371.

93 per cent. of the dried feces was fat, and with chronic pancreatitis the fat per cent. has been as high as 80 to 82.

With pancreatic disease, observers report *a great reduction in split fat.*

Fr. Müller[1] states that in normal feces from 20 to 30 per cent. of the fat is neutral fat, while from 70 to 80 per cent. is split fat, in the form of fatty acids and soaps. With *pancreatic disease,* the feces, on the other hand, may contain a *diminished proportion of split fat,* although the total fat may not be increased.

Fitz has collected 7 cases of pancreatic disease without jaundice in which there was no *steatorrhea.* The proportion of neutral fat was normal or less than normal in only 1 case, while in the rest it averaged 56.84 per cent.

One must remember that cases of undoubted pancreatic disease occur in *which there is no marked loss of fat,* so that the absence of *steatorrhea does not exclude pancreatic disease.*

The author has already referred to the fact that though disease of the pancreas is present, that even with closure of the duct of Wirsung, a patent duct of Santorini may allow the escape of pancreatic juice into the intestines.

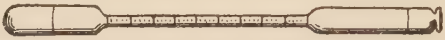

Fig. 363.—Schmidt-Stokes milk-tube (Robson and Cammidge).

Early in the stages of the disease, little change will occur in the fat-content of the stool, and if little fat be ingested by the patient or milk be taken, in which form the emulsified fat is readily absorbed, no noticeable difference would occur.

Some have also offered the theory that there is an increase in the activity of the stomach lipase.

One must consider all these possibilities, and hence *early diagnosis is often difficult and frequently impossible.*

In order to estimate the amount of fat in the stools, it is necessary to employ a test-meal containing known amounts of fat. One can employ the Schmidt-Strassburger test-diet or Steele's modification, as under Testing the Intestinal Functions, or that recommended by Brugsch, which consists of 2 liters of milk, 150 grams of white bread, and 50 to 100 grams of butter. With this meal a capsule of carmine is given to identify the resulting stool. It would seem best to divide this into two or even three meals.

The characteristics of a fatty stool have already been described.

Robson and Cammidge describe a special method of their own for determining the fat in the feces. Their technic is as follows:

"They employ two dry, clean Schmidt-Stokes milk tubes, which they label 'A' and 'B' (Fig. 363). Each is provided with a

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[1] Zeit. für Klin. Med., xii, p. 51, 1887.

10 c.c. mark. About ½ gram of finely powdered feces, which have been dried on a water-bath, is introduced into the lower bulb of each tube.

"The residue, on the watch-glass used for weighing and on the sides of the short funnel through which the powder is poured into the tubes, is washed down by a fine jet from a wash bottle, which for A tube contains hydrochloric acid (1 : 3), and for B tube, plain water.

"The sides of the tubes are also washed until all is collected in the lower bulbs, and the tubes are each filled with solution to the 10 c.c. mark.

"Tube A is then heated in boiling water for twenty minutes, rotating it occasionally so as to thoroughly mix its contents. After it is cooled, both tubes are filled to the 50 cc.. mark with ether. They are then tightly corked and inverted about forty times, so that all the material runs from bulb to bulb at each turn. Each tube is then rotated between the hands and then allowed to stand for half an hour or more, so that the solid residue may collect in the lower bulbs. The upper layers of ether in the tubes should be clear and free from solid particles.

"Draw off from each tube 20 c.c. of the ethereal extract with a pipet, and place each in a $CO_2$ flask of known weight, also noting the amount of ether left in the two tubes.

"The ether in each flask is then evaporated, the residue dried on a water-bath, and the flasks are again weighed.

"From the amount of extract yielded by the 20 c.c. of ether, and the quantity of ether left in the tubes, the total amount yielded by the weight of dried feces used may be calculated, and from this the percentage in the stool may be determined.

"The result from tube A gives the total fat in the feces, *i. e.*, neutral fats, free fatty acids, and combined fatty acids or soaps; since the latter are decomposed by boiling with hydrochloric acid and are thus rendered soluble.

"The residue from B tube represents the *neutral fats* and *fatty acids*, as the soaps are undissolved by the ether.

"The difference between the two gives the proportion of saponified fat. Other substances in the feces are negligible. For convenience we may say tube A gives the total fat, tube B, the 'neutral fat', and the difference between the two, the 'fatty acid.'

"Robson recommends testing the solid residue of tube B for stercobilin. The contents of B are filtered off, then extracted with acid alcohol, the extract neutralized with ammonia, and then mixed with a quantity of 10 per cent. zinc acetate in alcohol. The precipitate is removed by filtration and the clear filtrate examined through a lens against a black background.

"A green fluorescence indicates the presence of stercobilin. The intensity of the color varies with the amount of the pigment."

The total absence of pancreatic juice and bile, however, does not always put an end to the fat-splitting process in the intestines; by the organisms of the colon group fats are converted into glycerin and fatty acids in the lower part of the small intestine. Absorption will be diminished, and they will be largely excreted in the feces. The appearance of a larger proportion of saponified fat in the stools than would be expected in some cases of severe pancreatic disease is probably explained in this way.

The author has already referred to the influence of simple biliary obstruction, defective gastric digestion, excess of fat in the diet, disease of the intestinal mucosa, intestinal tuberculosis, tubercular peritonitis, etc., which would all influence the excretion of fat, and which must be excluded.

**Color of the Stool in Steatorrhea.**—The white appearance of the feces due to diminution or exclusion of the pancreatic secretion from the intestines can be attributed chiefly to the presence of excess of fat, especially the crystalline fatty acids. It is believed to be partly due to the reducing action of bacteria growing anërobically in an acid medium. Müller has attributed it chiefly to the absence of bile through the blocking of the biliary passage. When such occurs it would be a contributory factor, but it can be demonstrated that bile is present in some cases.

## THE USE OF PANCREATIC ENZYMES AS AN AID TO DIAGNOSIS; PANCREATIC INFANTILISM

In the case of animals in whom the pancreas had been removed, it was found possible to increase the assimilation of fats and proteins by the administration of fresh pancreas.

**Pancreatic Infantilism.**—Bramwell[1] describes a case in which he believed that retarded development was due to deficient pancreatic action. The patient, eighteen years of age, had suffered from diarrhea for nine years, and there was arrested development after the age of eleven. There were disappearance of diarrhea and a rapid increase in weight after the administration of a glycerin extract of the pancreas.

Thompson[2] and Rentoul[3] record similar instances, though Herter is inclined to attribute them to chronic intestinal putrefaction.

Robson and Cammidge report a case of chronic pancreatitis demonstrated by operation, in which after the administration of "pancreon" the neutral fat was diminished over 40 per cent., and the fatty acid increased 11 per cent. in the stools.

The use of *pancreatic extract might*, therefore, be of *some aid to diagnosis, especially if improvement in the stools, with diminished fat excretion, resulted therefrom.* This would suggest at once pancreatic deficiency.

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[1] Scottish Med. and Surg. Jour., 1904, xiv, 321.
[2] Robson and Cammidge.
[3] Brit. Med. Jour., 1904, ii, 1011.

**Azotorrhea.**—The presence of unaltered muscle-fibers in the feces is significant of disturbance of protein digestion.

Fitz[1] found only 8 cases in which this occurred in pancreatic disease. Examination should be made under the microscope, where the muscle-fiber is readily recognized (Fig. 364).

Rarely, undigested muscle can be detected in the stool with the naked eye. In forming proper judgment, one must exclude an excessive meat diet, and also cases in which for any reason there is increased peristalsis, as in intestinal catarrh, or intestinal putrefaction with secondary diarrhea.

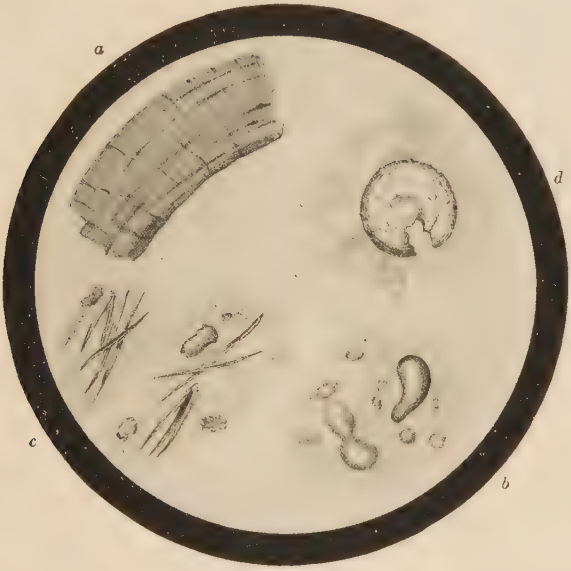

Fig. 364.—Microscopic characters of the residues met with in the stools in cases of pancreatic disease and biliary obstruction: *a*, Striated muscle-fibers; *b*, fat globules; *c*, free fatty acid crystals; *d*, combined fatty acid (soap) crystals (Robson and Cammidge).

In such event, the contents are hurried through the intestines before they can be digested. The stomach, moreover, digests the connective tissue of meat, and deficient gastric digestion would lead to imperfect digestion of muscle, since the meat fibers, under such conditions entering the intestines, bound together by connective tissue, would be attacked less rapidly by the pancreatic juice. Undigested meat fibers would thus readily pass through the intestinal canal.

**Nitrogen in the Feces.**—Under normal conditions only 5 to 6 per cent. of nitrogen is lost in the feces. In a case of pancreatic diabetes, Hirschfeld[2] recovered 32 per cent. of the nitrogen ingested with the food. In pancreatic disease without jaundice,[1] the loss of nitrogen is 21 per cent.; while it jaundice is present, it may reach 33 per cent.

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[1] Trans. Cong. of Amer. Phys. and Surg., 1903, vi, 36.

[2] Zeit. f. Klin. Med., 1891, xiv, 294.

## CARBOHYDRATES IN THE STOOL. PANCREATIC DIASTATIC FERMENTS

Though extirpation of the pancreas in animals causes disturbance of the digestion of the carbohydrates, so that an increased proportion of starch ingested reappears with the feces, Opie holds that the determination of the undigested carbohydrates in the human being acquires no significance for the diagnosis of pancreatic disease.

Robson and Cammidge find that though in some instances of pancreatic disease a larger proportion of carbohydrates than normal is found in the feces, this is not constant even in marked cases. The fact that the patient, in spite of an abundant carbohydrate diet, loses weight, points to a diminished assimilation of carbohydrates in excess of the conditions indicated by the feces, and hence the figures given by analysis of the stool are not the true index of the loss of carbohydrates to the organism.

The difference between the quantity assimilated and that found in the stools is probably explained by bacterial action.

The influence of a pathologic condition of the fecal flora (see Testing the Intestinal Functions) must also be considered. Hyperchlorhydria would be productive of fermentation of the carbohydrates in the stomach, and catarrh of the intestines would have an influence. One is, therefore, compelled to arrive at definite conclusions after a process of careful elimination.

Wohlgemuth[2] introduced the method of estimating the diastatic ferments in the feces.

Wynhausen[3] employs the following method:

"Five c.c. of a 1 per cent. solution of soluble starch (Kahlbaum) are each placed in several test-tubes of the same size, and graduated quantities of the filtrate of a thin stool are added to each. The tubes are placed in an incubator at 40° C. for twenty-four hours. At the end of this period they are filled to within a finger-breadth of the top with distilled water. To each is then added 1 drop of one-tenth normal solution of iodin and the resulting color is noted. If the starch is undigested, a blue color results; if erythrodextrin is present, it is red; if complete digestion has occurred (achroödextrin), the solution is colorless.

"To calculate the amount of ferment, if 1 c.c. of the filtrate digests 1 c.c. of soluble starch (Kahlbaum) in twenty-four hours at 38° C., the filtrate has a concentration of one diastatic unit. If one-tenth of 1 c.c. of the filtrate is added to 5 c.c. of the starch solution, and the mixture is colorless on adding 1 drop of the decinormal iodin solution, digestion has taken place. Therefore, 1 c.c. of the filtrate added to 1 c.c. of the starch solution equals 10 × 5 or 50 units."

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[1] Lyle, N. Y. Med. Jour., May 28, 1910.

[2] Zeitschr. für Biochemie, ix, 1908.

[3] Berlin. Klin. Woch., July, 1909, p. 1406.

Wynhausen believes the normal number of units should be about 500 to 600, or even up to 2000. If the total number of units is as low as 50 to 60, there is an interference with the flow of pancreatic juice. Lyle shows that considerable caution must be exercised in this estimation, as the units may be only 60, with a normal evacuation, and in the same case, after a dose of Carlsbad salts, be increased to 600. The method of producing the stool must, therefore, be considered.

Fedeli[1] and Romani describe the following method for estimating the diastatic function of the pancreas:

"To 1 c.c. of saliva add 5 c.c. of the patient's gastric juice. Shake the mixture, and in half an hour add sufficient of a 1 per cent. sodium carbonate solution to render it slightly alkaline. To this add 20 c.c. of a 10 per cent. starch paste and place in a thermostat at 37° C. for two hours, shaking it repeatedly.

"Estimate the amount of sugar formed.

"Then add 10 c.c. of an aqueous solution (1 : 4) of feces to the above, and leave in an incubator for twelve hours, when the sugar formed is again estimated. The difference between the two quantities of sugar represents the degree of pancreatic diastatic function."

## GENERAL CHARACTER OF THE STOOL

With *advanced* pancreatic disease the stools are frequently *bulky*, soft, white, and usually have an acid reaction and a peculiar odor. This appearance does not always occur, however. There is an abnormal quantity of undigested material, especially of fat, and also marked fermentation is present.

**Reaction of the Stool.**—Out of 80 cases of pancreatic disease reported by Robson and Cammidge, the fresh feces had an acid reaction in 58 patients; in 16 they were neutral or amphoteric, and in 6 they were alkaline.

Jaundice appeared to exert little effect on the reaction of the stools when the pancreas was also diseased. With jaundice without pancreatic disease, and in patients where calculi were present in the biliary passages, but no bile-pigment in the urine, the stools were usually alkaline.

The peculiar sour odor of the stools in pancreatic cases is probably due to the free fatty acids.

In the fatty stools of jaundice Strassburger noted a diminution in the number of bacteria, and probably there is a lessened degree of intestinal putrefaction in these cases, even though the bile is believed to possess antiseptic properties. If, however, the amount of protein is increased, putrefaction may occur as a result of affections of the pancreas or intestines, and the feces may become alkaline.

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[1] Riforma Medica, Sept. 20, 1909.

Enteritis or chronic colitis, which may be associated with pancreatic disease, may account in some for the alkaline reaction of the stool.

## CHANGES IN THE URINE IN PANCREATIC DISEASE

**Indicanuria.**—Opposing theories are held as to the relation of pancreatic disease to indicanuria.

Herter believes that since a reduced secretion of pancreatic juice is followed by an impaired digestion of proteins, the latter are liable to be attacked by bacteria, the urine showing the signs of intestinal putrefaction (indicanuria), holding that on exclusion of the bile and pancreatic juice there is an excess of indican, and that the ethereal sulphates are increased, their proportion to preformed sulphates rising to 1 : 6 or even 1 : 1; while the normal proportion is 1 : 10. Other observers believe that absence of indican and a diminution of ethereal sulphates are an indication of pancreatic disease. One may say that diminished secretion of the pancreas provides conditions under which putrefaction may occur.

Robson believes that the presence of indicanuria and an excess of ethereal sulphates in the urine probably shows an associated enteritis.

**Bile in the Urine.**—Diseased conditions of the pancreas, or cholelithiasis which may obstruct the passage of bile into the intestines, may produce jaundice, and the appearance of bile in the urine. The latter may in some cases be of a deep yellow or brown color, and gives the reaction for bile-pigment. Bilious urine and jaundice are not constantly found.

**Urobilin.**—If the bile is *completely shut off from the intestines*, no urobilin is found in the urine. When *urobilin is present in the urine*, there is some escape of bile into the intestines, even though there is an apparently complete obstruction of the duct.

**Azoturia.**—An increased excretion of nitrogen compounds in the urine is believed to result from disturbances of intestinal digestion, and to be closely related to putrefactive changes in the intestines. This condition is not constant in diseases of the pancreas, though it does occur with diabetes. In many cases of pancreatitis the excretion of urea is not excessive and the total nitrogen is within normal limits.

**Phosphates.**—These are stated by some to be increased in diseases of the pancreas, but there is probably no marked variation. This is true also of the chlorids.

**Acetonuria.**—Experimentally, extirpation of the pancreas in animals may result in the appearance of acetone, diacetic acid, and, at times, of $\beta$-oxybutyric acid in the urine. Acetone and diacetic acid have been found in the urine in a large percentage of cases of acute pancreatitis, and in nearly one-third of the cases of chronic pancreatitis and of malignant growth which have been examined for these bodies. Acetone bodies occur with *diabetes*, with wasting diseases, in postanesthetic poisoning, with gastro-intestinal diseases, with the cyclic vomiting of children, with the toxemic vomiting of pregnancy, with puerperal eclampsia, with phosphorus-poisoning, with acute yellow atrophy of the liver and other diseases of that organ, with fevers, with scurvy, starvation (especially in women and children), etc. Carbohydrate starvation may also produce acidosis. The condition is not idiopathic to pancreatic diseases or to pancreatic diabetes. It is now believed that the acetone bodies are formed chiefly from the storage fats within the system, and not within the intestinal tract.

**Calcium Oxalate.**—These crystals have been found in many cases of chronic pancreatitis, and diabetes has been occasionally observed to follow long-continued oxaluria. Part of the calcium oxalate is undoubtedly derived from the food, and *oxaluria may be the direct result of intestinal fermentation.* The writer believes it has *no significance* as to pancreatic disease.

**Carbohydrates.**—*Alimentary Glycosuria.*—The liver is limited in its ability to transform glucose into glycogen, and if an excess of sugar is absorbed from the alimentary tract the quantity of sugar in the blood is increased and is, consequently, excreted in the urine (alimentary glycosuria). A normal person can assimilate from 150 to 250 grams of glucose after fasting. Therefore, Kraus advises giving 100 grams of dextrose (grape-sugar) in 250 c.c. of tea or water two hours after breakfast, on the fasting stomach, and testing the urine for sugar two or three hours later. If this is found, there is a possibility of pancreatic disease, showing the metabolic function of the pancreas is at fault.

Temporary glycosuria occurs with a variety of injuries or operations on the nervous system, with neurasthenia, traumatic neurosis, some forms of mental debility, after the use of large quantities of carbohydrates, acute febrile conditions, alcoholism, with exophthalmic goiter, acute diseases of the brain and meninges, mania, paralysis, and, in some cases, with cirrhosis of the liver. It is present with asphyxiation, carbon monoxid poisoning, after an excessive dose of morphin, curare, etc. It shows pancreatic disease to be present in about 65 per cent. of cases. *Persistent glycosuria is suggestive of diabetes.* Fehling's test will disclose the presence of sugar.

**The Cammidge Pancreatic Reaction.**—This reaction consists in the demonstration in the urine, when treated by a special chemic procedure, of certain crystals of a definite morphology and a certain characteristic solubility. Their composition is unknown, according to Cammidge, though the substance on hydrolysis yields a body giving the reactions of pentose, probably an osazone. The specific reaction that is secured is believed to be due to an inflammation of the pancreas. It has also been found in a percentage of cases of carcinoma of the pancreas, due to an inflammatory zone about the neoplasm.

The "A-reaction" and, later, a differential test, the "B-reaction," were first employed. It was found that errors in technic were apt to occur, and that much depended on the individual equation. The "improved" or "C-reaction" is now employed.

The technic of C-reaction, as described by Cammidge, is as follows:

"The urine should be freed from albumin by boiling with acetic acid and from sugar by fermentation, when they are present. Then filter it several times through the same paper. A specimen of the twenty-four-hour urine should be employed, though the mixed night and morning specimen can be used.

"Forty c.c. of the clear filtrate are mixed with 2 c.c. of strong hydrochloric acid (sp. gr. 1.16), and the mixture is gently boiled on a sand-bath in a small flask which is fitted with a funnel condenser. The flask is cooled in a stream of water and the contents made up to 40 c.c. with cold distilled water. Then 8 grams of lead carbonate are slowly added to neutralize the excess of acid, and the solution should be allowed to stand a few minutes. It is again cooled in running water and filtered through a well-moistened and close-grained filter-paper until the contents are perfectly clear. Several filtrations may be required.

"The acid filtrate is shaken well with 8 grams of tribasic lead acetate and the resulting precipitate filtered out. A clear filtrate should be secured, filtering being performed several times if necessary.

"The excess of lead in solution is removed by a stream of sulphuretted hydrogen or, preferably, the filtrate is shaken well with 4 grams of powdered sodium sulphate, and the mixture then heated to the boiling-point. It is then cooled to as low a temperature as possible in a stream of running water and the white lead sulphate precipitate is filtered out.

"Ten c.c. of the clear, transparent filtrate are diluted up to 17 c.c. with distilled water, and to it are added 2 grams of sodium acetate, 0.8 grams of phenylhydrazin hydrochlorate, and 1 c.c. of a 50 per cent. acetic acid. This is placed in a small flask with a funnel condenser and boiled in a sand-bath for ten minutes. It is filtered while hot through a small filter-paper moistened with hot water into a test-tube provided with a 15 c.c. mark. If the filtrate falls short of the 15 c.c. mark, it is made up to this point by the addition of hot distilled water, and the mixture is stirred with a glass rod. In well-marked cases of pancreatic inflammation a light yellow flocculent precipitate should appear in a few hours, but in less marked cases it may be necessary to leave the preparation over night."

Under the microscope the precipitate is found to consist of long, flexible, light yellow and hair-like crystals, arranged in delicate sheaves (Fig. 365), which disappear in ten to fifteen seconds when irrigated with a 33 per cent. solution of sulphuric acid *A microscopic examination must always be made.*

Cammidge recommends a control experiment to exclude traces of sugar which may be undetected by the ordinary tests. He treats 40 c.c. of urine in the same way as just described, only that it is not boiled with hydrochloric acid.

The urine should be fresh, and if alkaline should be acidified with hydrochloric acid before testing.

Dextrose, when present, should be removed by fermentation *after* the urine has been boiled with the hydrochloric acid, and the excess neutralized. If *calcium chlorid* has been given, which is advocated in all pancreatic cases before operation, it *interferes with the test.*

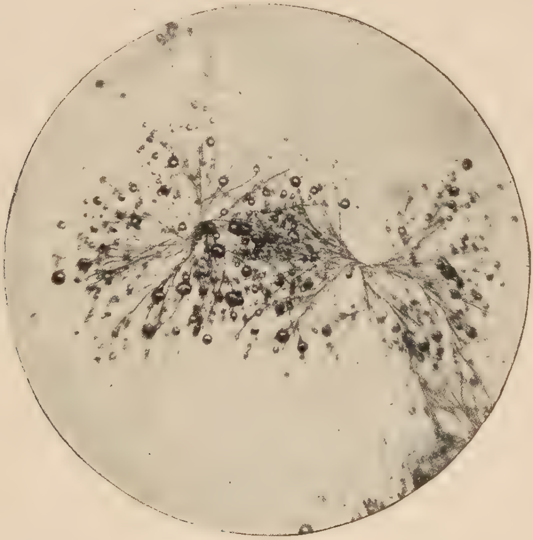

Fig. 365.—Improved or C- ("pancreatic") reaction crystals from a case of chronic pancreatitis with gall-stones in the common bile-duct (× 200) (Robson and Cammidge).

Robson and Cammidge[1] report the reaction positive in every case of acute and chronic pancreatitis—67 cases in all; and that in 16 cases of carcinoma it was found in 4 (25 per cent.). In 50 normal cases it was absent, while in 117 cases of gall-stones, etc., with no pancreatic disease, it was present only four times. In 75 per cent. of diabetics there was a positive result.

Opie[2] states that the pancreatic reaction has been obtained in a large number of cases in which pancreatic disease has been demonstrated or suspected; but the reaction may occur *without* pancreatic disease and is sometimes *absent when a lesion of the gland is present.*

[1] The Pancreas.

[2] Disease of the Pancreas, 1910.

Schumm[1] reports unfavorably, while Ochsner believes that further development of the test is necessary.

Kreinitz[2] found the results were in accord in 80 per cent. of his operative findings, though positive reactions occurred in diseases of the intestinal tract, biliary tract, diabetes, and in carcinoma of other organs than the pancreas.

Deaver[3] reports 154 reactions, and, more recently, 197 additional cases, and now summarizes 351 in all.[4]

In all the cases in which the condition of the pancreas was accurately determined by Deaver at the time of operation, the pancreatic reaction was obtained, on an average, only two and a half times as frequently when the pancreas was affected as when it was not. He holds that there is a degree of dependance for this reaction in pancreatic disease, but that it is not as intimate as we had been led to hope. More recently he finds only about 25 per cent. positive reactions corresponding to operative findings.[5]

Speese[6] and Goodman found the reaction positive in cases of injury of the pancreas or in hemorrhagic pancreatitis, but inconstant in subacute pancreatitis.

Swan[7] and Gilbride believe it an error to interpret every positive reaction as indicative of organic disease of the pancreas.

Roper and Stillman[8] carried out a series of studies on the Cammidge reaction, and found that results varied in the same patient, apparently due to accidental occurrences.

They conclude that the C reaction of Cammidge does not rest on a sound scientific basis.

The author has tested the reaction in a number of patients. In a recent case, for example, of far-advanced chronic pancreatitis with marked steatorrhea, repeated examinations showed a negative Cammidge. He must confess that he has been disappointed in his results secured as an aid to diagnosis. He still employs it as routine to further test it, though *skeptical* as to its value.

**Other Urinary Tests in Pancreatic Disease.**—Wohlgemuth has advised estimating the diastatic ferment in the urine as a means of diagnosis of pancreatitis.

He suggests in a suspected case that the urine should be examined daily, and a decided increase in the diastatic ferments should be regarded as indicating an interference to the flow of the pancreatic juice.

If 1 c.c. of urine digests 1 c.c. of soluble starch (Kahlbaum) in twenty-four hours at 38° C., this urine has a diastatic concentration of one diastatic unit.

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[1] Munch. Mediz. Wochenschr., 1909, Nos. 37 and 40.

[2] Arch. f. Verdaungskr., 1909, xv, 53.

[3] Amer. Jour. Med. Sci., Dec., 1910.

[4] Jour. Amer. Med. Assoc., April 15, 1911.

[5] Ibid., July 1, 1911.

[6] Amer. Jour. Med. Sci., 1909, cxxxviii, 108.

[7] N. Y. Med. Jour., 1910, xci, 781, 852.

[8] Arch. of Int. Med., Feb., 1911, pp. 252–258.

The formula for $y$ c.c. of urine and 5 c.c. of starch is $\frac{1}{y\ \text{c.c.}} \times 5$ (1 c.c. starch) = diastatic unit.

*Fat-splitting Ferment.*—Opie[1] found in 1 case of acute pancreatitis with fat necrosis evidence of a fat-splitting enzyme in the urine.

Hewlett[2] has also demonstrated experimentally that an injury to the pancreas may cause a fat-splitting enzyme to appear in the urine. In subacute and chronic pancreatitis Robson and Cammidge found no fat-splitting enzyme present.

**Lipuria.**—Bowditch and Clark describe the appearance of fat-globules in the urine in cases of cancer of the pancreas. They have also been found in acute pancreatitis, and Robson reports them in 1 case of chronic pancreatitis. Lipuria at times occurs with diabetes mellitus. It has been found in other conditions and is not diagnostic of pancreatic disease.

## THE BLOOD IN DISEASES OF THE PANCREAS

In the more chronic and more advanced cases there is generally a secondary anemia, with reduction in the red cells. The hemoglobin does not seem to suffer a proportional decrease with the red cells and the hemoglobin index may even be quite high. Often the ratio of the leukocytes is unaltered, though Woolsey has reported leukocyte counts from 17,600 to 39,000 in 3 cases of acute pancreatitis. The polynuclears should also be determined.

**Pancreatic Hemolysin.**—Friedeman[3] has demonstrated the presence of a strongly active autohemolysin in the pancreatic juice, and that when this enters the circulation through tissue destruction it brings about the same results as other hemolytic substances. In some pancreatic affections there is a tendency to general hemorrhage; thus, in cancer of the head of the pancreas, in pancreatitis with jaundice (believed to be partly due to cholemia), and also in pancreatitis without jaundice.

**Coagulation of the Blood.**—The *coagulation time* of the blood is *increased.* This feature is of considerable importance, as severe postoperative hemorrhage may occur in cases in which the pancreas is involved. The writer believes *the determination of the coagulation time of the blood may in some cases be of assistance in diagnosis.*

**Conclusions.**—The author would first recommend that in *every case* of suspected pancreatic disease the *functions of the stomach,* both *motor* and *secretory,* should be examined, so that the digestive function of this organ can be *taken into consideration* in making one's deductions.

The *general test of the intestinal functions,* as described on pages 492 to 500 by the Schmidt-Strassburger diet, should *then be made.*

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[1] Johns Hopkins Hosp. Bull., 1902, xiii, 117; also Kastle and Loevenhart, Amer. Chem. Jour., 1900, xxiv, No. 16.

[2] Jour. of Med. Research, 1904, xi, 377.

[3] Deut. Mediz. Wochenschr., 1907.

This demonstrates *whether or not a catarrhal condition of the intestine is present*, the presence or absence of bile, the influence of the fecal flora, fermentation and putrefactive conditions, and a general idea of the quantity of fat present, muscle-fibers, starch, etc., by microscopic examination. This alone will give pretty thorough information as to disturbance of the pancreatic functions. *Reduction of split fat is significant.*

For the *general practitioner*, the Boldyreff method of securing the pancreatic juice is the most *simple* and is to be *recommended*. Tests can then be made for trypsin, amylopsin, and steapsin.

The *urine should always be examined for sugar*. The Cammidge reaction may be tested for as routine, though its value is *doubtful*. Improvement in the *fecal findings after administration of pancreatic extract is also suggestive*. The *blood examination* should *be made* and the rapidity of blood-clotting determined.

These methods of examination, when employed in association with the clinical symptoms, will be of most service. In acute conditions particularly, and even with suspected chronic pancreatitis, the urine should be examined for colon bacilli, and if such are present, proper treatment instituted. The writer later reports a *chronic pancreatitis with colon bacillus infection*. The other methods described are of chief interest to the specialist.

## CHAPTER XXXVIII

# GENERAL SYMPTOMS AND DIAGNOSIS OF PANCREATIC DISEASE

One might suppose that on account of the important part that the pancreas takes in the digestive processes in the intestines, and its influence on the metabolism of the body, that any deviation from the normal would produce such functional disturbances that a diagnosis of pancreatic disease would be easy. On account of the fulminating symptoms, an acute disturbance of the pancreas is much more readily diagnosed than the chronic conditions. Unfortunately, the pancreas is seldom diseased without the involvement of some of the other organs. Thus, cholelithiasis, gastro-intestinal catarrh, or catarrh of the biliary and pancreatic ducts may coexist; or tumors or ulcers of the stomach or duodenum may involve the pancreas. Affections of the liver, lymphatic glands, or colon may also produce disease of the pancreas.

One must further remember that the stomach can care for the proteins to a certain extent, that the salivary and intestinal glands can digest starches, and that the bile and intestinal juice can emulsify fat; furthermore, the intestinal bacteria can break down various food stuffs, and thus interfere with the pathologic changes in the stools, such as would be expected in disease of the pancreas. When the duct of Wirsung is obstructed, in a number of cases the pancreatic secretion may escape through the duct of Santorini, and, moreover, a considerable portion of the pancreas may be diseased, and yet a sufficient part remain healthy and so carry on the functions of the organ. Sometimes, though the pancreas may be primarily at fault, the most prominent symptoms may be produced by an organ that is secondarily involved. For example, marked jaundice and distention of the gall-bladder may result from a cancer of the head of the pancreas pressing on the common bile-duct, or from pressure contraction of a chronic pancreatitis.

Moreover, as in a recent case of the author's, a pancreatic tumor may compress the intestines and produce *intestinal obstruction,* or may press upon the neighboring ganglia and cause such excruciating pain that it mimics that of an aneurysm or of spinal disease.

All these factors must be held in consideration.

**Anamnesis.**—*A careful history of the case is of great importance.* Some patients may give a history of habits of eating or drinking which might set up an inflammation of the stomach and duodenum; in others there may be a history of infectious conditions which are liable to be followed by disease of the biliary passages; or there may be a preceding history of disease of the bile-ducts or gall-bladder, with, in some cases, attacks of biliary colic. Males, moreover, seem to be more liable to pancreatic disease.

## PHYSICAL SIGNS

**Presence of Tumor.**—The position of the pancreas, lying behind the stomach, renders it unfavorable for palpation, and often, especially in stout persons, it cannot be felt at all. If the patient is very thin and the stomach is empty and the abdominal muscles are relaxed, especially in the case of gastroptosis, it may be often defined. Frequently in acute and chronic inflammation, and even in the case of abscess of the pancreas, it is impossible to determine enlargement of the pancreas. In the acute cases, pain and rigidity in the epigastrium often interfere with the determination of a mass, but the *acute fulminating attack* and the *epigastric peritonitis* are themselves suggestive. Robson claims that in many cases a distinct swelling can be felt due to the enlarged pancreas, with an effusion of blood, inflammatory fluid, and matted omentum.

Körte describes 3 cases in which the mass was palpable between the stomach and colon.

With chronic pancreatitis, in 3 cases only, in very thin patients, was Deaver[1] able to palpate the swollen head of the pancreas. Palpation he found of most value to *exclude other abdominal conditions*.

With cancer of the head of the pancreas, the only tumor usually felt is due to the enlarged gall-bladder.

Occasionally, tumor of the body, tail, and, at times, some growths of the head of the pancreas can be distinguished by palpation. The writer has recently determined one such by palpation.

By inflating the stomach with carbonic acid gas or with air the relation of this organ to the tumor can be made out. The inflated stomach separates the mass from the liver. Unless the stomach is empty, there is resonance on percussion over the growth, with communicated non-expansile pulsation, and a slight movement on deep inspiration. Inflation of the colon may also aid at times in localizing the mass. With cyst of the pancreas, the *presence of a tumor* may be for a time the *only symptom*. Its position depends on from what part of the pancreas it originates. It is found in various situations, which are described under Cyst of the Pancreas. *The absence of tumor does not prove that disease of the pancreas is not present.* If the tumor or enlargement of the organ can be *positively determined*, in *connection with other symptoms*, it affords *valuable evidence of a lesion* of the pancreas.

**Pain.**—Pain in acute pancreatitis is one of the *important symptoms*. It appears *suddenly* and is *intense in character*. It may be colicky, then diminish and disappear, and return later (paroxysmal). It may at times be continuous. It is usually located in the *epigastrium*, above the umbilicus, and may pass from the median line along the course of the pancreas.

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[1] Jour. Amer. Med. Assoc., April 15, 1911.

Opie notes cases in which the pain is most severe in the hypochondriac regions or even below the umbilicus.

In the cases of chronic pancreatitis reported by Deaver[1] *pain* was the *leading* and most *constant* symptom, being absent in only 3 cases. It varied from a dull discomfort, fulness, or oppression in the epigastrium to an ache or a sharp, lancinating, colicky pain, much like gall-stone colic. In the cases of colicky pain he found the gall-bladder usually diseased.

In many cases of chronic pancreatitis, especially in the *early stages*, *pain may be absent or scarcely noticed.*

There is no definite relation to eating or to particular articles of food in the production of the pain. In this it differs *from the pain* produced with *gastric or duodenal ulcer*, and thus may be of some aid in differential diagnosis.

The carbohydrates in some cases seem to cause the most digestive disturbances, and Sailer has found that the administration of glucose to test the limit of assimilation to be particularly distressing. Intestinal fermentation is undoubtedly the cause of this feature.

With malignant disease of the pancreas, *pain may be absent in some cases and present in others.* The pain may be very severe, due to pressure on or involvement of the ganglia or adjacent organs, such as the stomach and duodenum. On the other hand, the writer has recently seen a large carcinoma of the outer third of the pancreas, involving the stomach also, in which all symptoms pointed to gradually increasing stenosis of the bowel. Operation demonstrated pressure stenosis of the transverse colon. The patient had no pain from the tumor, which was readily palpable, and only suffered from cramps when attempting to have a bowel action, with dyspeptic disturbances and progressive loss of weight.

Pancreatic cysts are sometimes painless, though pain is present in some cases.

With abscess of the pancreas, pain is frequently present, but not invariably so.

Calculus of the pancreas may exist for years without any pain. If the calculus progresses to the orifice of the pancreatic duct or becomes impacted in the ampulla of Vater, severe paroxysmal pain may occur which resembles gall-stone colic, and jaundice may be associated. The pain may become continuous. It may occur in the epigastrium or radiate to either side of the thorax.

With disease of the pancreas, the pain may *radiate to the left*, to between the scapulæ or under the left scapula. When it radiates to the right it resembles gall-bladder pain. The pain may also radiate to the cardiac region and resemble that of angina pectoris. Pain can scarcely be called pathognomonic of any particular type of pancreatic disease, except in the case of acute pancreatitis.

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[1] Jour. Amer. Med. Assoc., April 15, 1911.

**Tenderness.**—With acute pancreatitis there is excessive tenderness on pressure in the epigastric region, most marked at Robson's point (Fig. 366), above and to the right of the umbilicus, which is accompanied by muscular rigidity of the recti. At times the tenderness may extend along the course of the pancreas, and Fitz has

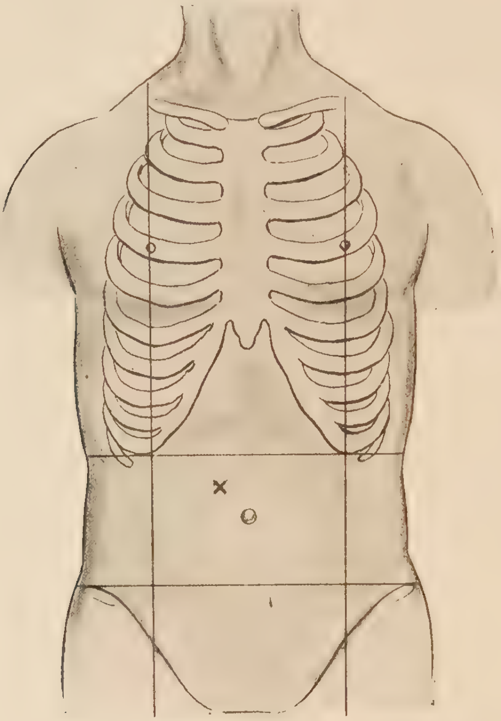

Fig. 366.—Most frequent site of the tender spot in inflammatory affections of the pancreas (" Robson's point").

noted additional sensitive points in the abdomen, probably due to the areas of fat necrosis.

With *chronic pancreatitis* tenderness may be absent, though in the more advanced cases it may be present. Deaver's patients were operated upon during or just after some exacerbation of the symptoms, and there was some degree of tenderness in most of them. His findings were variable as to location, tenderness being present below both *costal margins, more frequently to the right, in the mid-epigastrium, at Robson's point, and in one case general.*

**Rigidity.**—Rigidity was also variable, there being more cases in the right hypochondrium, some in the midepigastrium, and in some there was moderate distention.

Tenderness is, as a rule, absent in malignant disease of the pancreas, unless from pressure or involvement of the nerve plexuses or of adjacent organs.

With pancreatic abscess tenderness is often present, but it may be absent, and with pancreatic cysts tenderness is more usually absent.

With calculus of the pancreatic duct tenderness is often absent, unless the calculus becomes impacted in the ampulla, when tenderness may be associated with the attacks of paroxysmal pain.

## SYMPTOMS

**Nausea and Vomiting.**—With acute pancreatitis (hemorrhagic or gangrenous) *vomiting is a prominent symptom.* It is frequently *so violent and obstinate as to suggest intestinal obstruction.* The vomiting is *not stercoraceous*, and there is *no visible peristalsis of the intestines*, such as occur with acute obstruction.

In other types of pancreatic disease, vomiting has usually been considered not a common symptom, and when it occurs it is often due to disturbances of the stomach or duodenum.

Opie reports 4 cases of persistent vomiting associated with chronic interlobular pancreatitis. The history in all afforded evidence of gastric or gastro-intestinal disease, and indicated some relationship between these disturbances and the chronic lesion of the pancreas.

Deaver[1] reports 21 cases of chronic pancreatitis having attacks in which vomiting figured, and 10 more who were nauseated, and holds that in the more severe types of chronic pancreatitis nausea or vomiting may occur in attacks at variable intervals. When vomiting occurs, it is not particularly characteristic, though it may contain mucus and bile. If an abscess of the pancreas has ruptured into the stomach, pus and altered blood may be vomited up.

**Vomiting of Blood.**—With *acute pancreatitis*, altered blood—"*coffee-ground*" or so-called "*black*" *vomit—may be vomited up.* This may occur quite early in association with the tendency to general hemorrhage. The vomiting of *blood with this hemorrhagic diathesis* occurs chiefly with acute pancreatitis, in *pancreatitis associated with jaundice*, and also in *cancer of the pancreas*, though also in other types of pancreatic disease.

**Jaundice.**—Acute hemorrhagic pancreatitis may be accompanied by a slight jaundice if a small gall-stone is impacted in the outlet of the ampulla of Vater. There is a well-known relation between gall-stone disease and disease of the pancreas. The relation of the common bile-duct to the head of the pancreas is the determining factor in many cases as to the appearance or absence of jaundice; in about 38 per cent. of cases the common duct lies behind the head of the pancreas, so that pancreatitis, either acute or chronic, or even cancer of the pancreas, may run its course without the appearance of jaundice. In about 62 per cent. of cases the common duct is situated in a deep groove or is imbedded in the head of the pancreas, so that either pancreatitis or a new growth will compress the bile-duct and lead to jaundice.

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[1] Jour. Amer. Med. Assoc., April 15, 1911.

Robson reports that in 62 per cent. of cases of chronic pancreatitis with cholelithiasis bile-pigments were found in the urine before operation, while in 38 per cent. neither jaundice nor bile-pigment was present. In the case of chronic pancreatitis without cholelithiasis in 16 per cent. there was jaundice, due either to an ascending catarrh of the duodenum or to compression of the common duct.

In Deaver's patients with chronic pancreatitis jaundice was present in 24 and absent in 14. Jaundice may be preceded by pain, as in gall-stone colic, though usually it is less severe, unless a stone is passing through the duct.

Jaundice is not a constant symptom. *Marked jaundice* with a *distended gall-bladder* is significant of *cancer of the head of the pancreas.* Jaundice in these cases develops slowly but steadily, and usually without pain. The patient's skin, as the cachexia increases, may become of a slaty appearance, a "black jaundice," instead of the saffron yellow of cholelithiasis. With advanced chronic pancreatitis jaundice may occur. When a *gall-stone in the common duct* is the cause of the jaundice, the *gall-bladder is usually contracted*, and often cannot be felt (Courvoisier's rule).

**Emaciation.**—Emaciation as a symptom does not figure in acute inflammation of the pancreas, since the attack is fulminating in character and of short duration. In the typic chronic inflammation, in carcinoma or cyst of the pancreas, and in diabetes of pancreatic origin, emaciation is often a prominent symptom. The loss of weight is frequently marked. Due to disturbance of the pancreatic function, digestion is impaired and rapid loss of weight may result. It is sometimes difficult to differentiate between chronic pancreatitis of a severe type, with rapid loss of weight, and carcinoma of the pancreas.

Loss of strength may accompany the loss of weight. This is particularly true in cases of severe chronic pancreatitis with associated jaundice, and also in cancer of the pancreas.

**Temperature.**—In acute hemorrhagic pancreatitis the temperature may be normal or subnormal; while with acute gangrenous and acute suppurative pancreatitis the temperature may be considerable. In the last, it is frequently irregular and may run a septic type. With acute pancreatic catarrh the temperature resembles that of infective cholangitis. With abscess formation there may be a persistent temperature of 100° to 103° F., with rigors associated. With cancer of the pancreas the temperature is usually subnormal, though occasionally fever from associated complications may result. Cysts and calculus, as a rule, are not accompanied by fever.

With chronic pancreatitis the temperature, *as a rule, is normal,* but fever may occur during the exacerbation of the disease.

**Dyspepsia and Disturbance of Appetite.**—There may be anorexia, a sensation of fulness after eating, flatulence with eructations, heartburn, nausea, and distaste for fats and meat. Sometimes the administration of pancreatic extracts markedly relieves these symptoms.

**Hemorrhage.**—Reference has already been made to the hemorrhagic tendency which is found with inflammatory disease and malignant growths of the pancreas. Bleeding may occur, not only at and after operation, but at other times, and may manifest itself by hemorrhage from the mucous surfaces, stomach, intestines, etc., and into the skin or subcutaneous tissues.

**Blood.**—The blood frequently shows a secondary anemia, and leukocytosis may occur with the acute attacks. Clotting may be delayed.

**Pressure Symptoms.**—With disease of the pancreas ascites may at times occur due to tumor pressure on the portal vein, and edema of the lower limbs from pressure on the inferior vena cava. Enlargement of the spleen and hemorrhoids may also result from pressure on the portal vein. Occasionally the duodenum is partially or completely surrounded by the head of the pancreas, and malignant disease or inflammation of the pancreas may obstruct the passage of the gastric contents and produce dilatation of the stomach with vomiting.

Cysts or new growths of the pancreas may displace the stomach, duodenum, or colon. The stomach, depending on the position of the cyst, may be pushed up under the diaphragm or down below the umbilicus.

Distention of the gall-bladder with associated jaundice may result from cancer of the head of the pancreas, and also from chronic pancreatitis, when the common duct passes through the head of the organ. Occasionally the hepatic duct is pressed upon by a prolongation of the diseased pancreas, so there may be jaundice without distention of the gall-bladder. If a pancreatic cyst presses upward against the diaphragm, it may cause dyspnea, and this may also be produced by secondary effusion into the lesser peritonial sac. Hydronephrosis may occasionally be caused by pressure of pancreatic tumors on the ureter, and either pressure on or involvement of the solar plexus may cause pain.

**Liver.**—The liver is occasionally enlarged (cirrhosis), with diabetes.

**General Circulation.**—Symptoms of severe shock occur with acute pancreatitis, and the pulse becomes so rapid and feeble that cyanosis results.

**Bowels.**—With acute pancreatitis there is obstinate constipation. With pancreatic disease of a chronic type, constipation is a frequent symptom; rarely, diarrhea. The frequent soft, bulky movements, greasy and of pale color, described as characteristic of pancreatic disease of the more chronic type, are found in the *most advanced cases* and *not* in the earlier stages of the diseased pancreas.

**Steatorrhea.**—The microscopic appearance of fat in the stools is a valuable adjunct in determining disease of the pancreas. One must determine whether the clay-colored stool is due to the presence of excess of fat or fatty acids, or to absence of bile from the intestinal tract. The presence or absence of stercobilin in the stool will determine this last feature. In health the unabsorbed fecal fats consist approximately of 20 to 30 per cent. neutral fat, and from 70 to 80 per cent. of split fat (fatty acids and soaps). In cases of pancreatic disease, even though the total percentage of fecal fat in some cases may not be increased above the normal, *the ratio of split fat to the neutral fat is always decreased.* Katz has asserted that a diminution of the split fat below 70 per cent. of the total fecal fat signifies disease of the pancreas, except in nursing infants and patients with diarrhea. Robson and Cammidge hold that in chronic pancreatitis the total amount of unabsorbed fat may reach 50 to 60 per cent., and even to 75 to 90 per cent., with malignant disease of the organ.

Normal stools have, however, been found with cases of pancreatic disease, so that *negative findings* as to steatorrhea or split fat may *not be conclusive.* Positive results are *very suggestive.*

**Azotorrhea.**—The presence of muscle-fibers in the feces is not as constant a symptom.

**Sialorrhœa Pancreatica.**—An increased flow of saliva has been noted by a few observers in disease of the pancreas, especially in cases of pancreatic calculi and in cysts. As an aid to diagnosis it cannot be relied on.

Glycosuria should be tested for.

The author has recommended the most practical tests for diagnosis at the end of Chapter XXXVII, Methods of Diagnosis in Pancreatic Disease.

**Fat Necrosis.**—The discovery of fat necrosis by the surgeon on opening the abdomen should be an indication for exploration of the pancreas.

## CHAPTER XXXIX

# INJURIES OF THE PANCREAS—CLASSIFICATION OF ACUTE AND CHRONIC INFLAMMATION OF THE PANCREAS—CATARRH OF THE PANCREAS

### INJURIES OF THE PANCREAS

On account of its relation to other important viscera, it is rare for the pancreas *alone* to be damaged by direct violence without associated injury to other organs.

Injuries of the pancreas comprise laceration due to direct violence, bullet wounds, and penetrating wounds.

**Laceration Due to Direct Violence.**—In the majority of cases the force which produces the injury has been directed from before backward in the epigastrium, and lacerations of other organs, such as of the liver, kidney, duodenum, jejunum, spleen, or fractured ribs, have been associated. In such cases the hemorrhage from the other injured organs has recently been so severe that the injury to the pancreas has often been overlooked. Rarely, the pancreas alone may be lacerated.

*Etiology.*—Falls, a blow, a kick, crushing between two vehicles, or the passage of a vehicle over the abdomen are the most frequent causes of laceration. Sometimes the injury (blow) may be apparently slight.

*Symptoms.*—In some cases after severe injury, shock and the immediate symptoms of internal hemorrhage may occur, and operation will alone determine the character of the injury. In others, there may be only a slight hemorrhage into the pancreas as a result of the injury, and shortly acute pancreatitis follows; or the patient may slowly recover from the blow, shock pass off, and within a few days to a few days to a few weeks an abdominal tumor may appear. In this event laceration of the posterior layer of the lesser peritoneal sac has occurred, and blood and the pancreatic secretion are poured into the lesser peritoneal cavity; adhesions close the foramen of Winslow and a pseudocyst of the pancreas results.

It occupies the epigastric, umbilical, and left hypochondriac regions, as a rule. The stomach and transverse colon usually lie in front, and the descending colon behind and to the left.

*Treatment.*—The immediate shock should at first receive treatment, and as soon as reaction occurs, immediate laparotomy should be undertaken and an attempt be made to secure the bleeding points. Suture of the torn pancreas should be performed. If other viscera are lacerated the condition is complicated, but repair should be undertaken.

In cases of acute pancreatitis following hemorrhage due to traumatism, incision and drainage of the pancreas are indicated, and when a pseudocyst of the pancreas forms subsequent to injury it should be incised and drained.

**Bullet Wounds of the Pancreas.**—In the majority of instances other viscera have also been perforated as well as the pancreas. President McKinley, for example, was wounded in the stomach, pancreas, and left kidney. Rarely, the lesser omentum and pancreas may alone be injured.

*Symptoms.*—There are no symptoms pathognomonic to a wound of the pancreas. The probable course of the bullet is the only guide. When the stomach and lesser omentum are found to be wounded, particular examination of the pancreas should be made. The only symptoms are of shock and hemorrhage taken in association with the visible perforating wound.

*Treatment.*—Shock should be treated. Operation is indicated. Bleeding points should be tied. The injured organ should be sutured and proper drainage instituted.

**Penetrating Wounds of the Pancreas.**—Penetrating wounds of the pancreas have been reported as a result of stabs with a knife or bayonet.

In some cases the pancreas protruded, and was either returned or the projecting portion resected. These cases recovered. Injuries of other viscera were associated.

*Symptoms.*—They consist of shock and internal hemorrhage.

*Treatment.*—This is the same as that described for gunshot wound.

As a result of injuries to the pancreas, acute pancreatitis (hemorrhagic), a pseudocyst of the pancreas, a true pancreatic cyst, abscess of the pancreas, protrusion of the pancreas, and prolapse of the pancreas may occur.

## CLASSIFICATION OF ACUTE AND CHRONIC INFLAMMATION OF THE PANCREAS

Undoubtedly, inflammatory affections of the pancreas are much more frequent than were formerly supposed. We owe much to the surgeons who have demonstrated the frequency of pancreatic disease, and its particularly close association with disease of the gall-bladder.

As far back as 1672 Tulpius described a diffuse abscess of the pancreas, and Baillie, in 1799, a hard pancreas, evidently a case of chronic interstitial pancreatitis. In 1804 Portal described a case of acute suppurative pancreatitis, and Percival, in 1818, one of pancreatic abscess with jaundice. In 1879 Balzer first described acute pancreatitis with fat necrosis. Fitz first placed inflammatory diseases of the pancreas on a practical basis, and Robson, chronic pancreatitis and the relation of the pancreas to gall-stone disease.

**Classification.**—From a pathologic standpoint the writer believes that Robson's classification of pancreatic inflammation is the best:

(1) Catarrhal inflammations—acute, chronic, suppurative, and pancreolytic catarrh.

(2) Parenchymatous inflammations comprise acute hemorrhagic pancreatitis: ultra-acute, in which profuse hemorrhage precedes inflammation; acute, in which inflammation precedes hemorrhage; gangrenous pancreatitis; suppurative pancreatitis (diffuse or circumscribed). Chronic inflammation comprises interstitial pancreatitis of the interlobular or interacinar type, and cirrhosis of the pancreas.

**Etiology of Pancreatitis.**—Among the predisposing causes of pancreatitis are obstruction in the ducts or papilla of Vater resulting from gall-stones, duodenal catarrh, pancreatic calculi, cancer of the head of the pancreas or of the papilla, ulcer of the duodenum with cicatricial stenosis of the papilla, and lumbricoid worms. Injuries to the epigastrium or a wound may produce pancreatitis. Among other predisposing causes are hemorrhage into the pancreas, general diseases, such as typhoid, influenza, and mumps, anatomic peculiarities in the pancreas or its ducts, atheroma or fatty degeneration of the blood-vessels, back pressure from diseases of the heart and lungs, and new growths, notably cancer.

Among the exciting causes are infection from the blood, as from syphilis or pyemia, or extension from the duodenum, as with gall-stone obstruction or from gastroduodenal or duodenal catarrh; or from adjacent organs, as from gastric or duodenal ulcer, or cancer of these viscera eroding the pancreas.

Tuberculosis may be a cause of infection, and alcohol may be productive of pancreatitis, though such has been disputed.

Bacterial infection has been disputed in its causal relation to pancreatitis, though colon bacilli, streptococci, staphylococci, etc., have been found, and are believed by many to be secondary invaders of injured tissue. The author particularly believes the colon bacillus to be a factor in some cases, either from a general infection or through the duodenum, and later reports a case of advanced chronic pancreatitis with colon bacilluria.

Since the ducts of the pancreas open into the duodenum, which always contains bacteria, it is evident that infection can readily occur from this viscus. The close relation of the pancreatic to the common bile-duct is also a prominent factor. With the lodgment of a calculus in the common bile-duct, an infective cholangitis may follow. If a stone passes down into the pancreatic portion of the common duct, the duct of Wirsung may be compressed, and the secretion of the pancreas may be dammed back. This retained secretion may, in turn, become infected, and a catarrhal inflammation of the pancreas result. Even if the stone ultimately passes, the congested and swollen pancreas may keep up pressure on the common duct and cause a persistence of the jaundice.

With persistent jaundice, due supposedly to extension into the common bile-duct from a duodenal catarrh, probably the pancreatic duct is involved in many cases.

With persistent obstruction from a gall-stone a suppurative inflammation of the pancreatic and common bile-ducts may ensue, and if this continues, abscess of the liver and pancreas may follow.

With the milder type of infective catarrh an interstitial pancreatitis may result. This type is usually of the interlobular variety, but later the islands of Langerhans may become affected and diabetes result.

If the opening of the ampulla of Vater into the intestine is obstructed by a gall-stone (Fig. 367) the common bile-duct and duct of Wirsung become a single channel, and acute hemorrhagic pancreatitis may result from the entrance of bile into the pancreas.

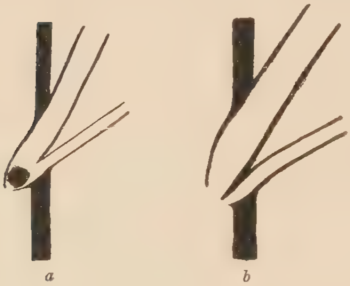

Fig. 367.—*a*, Diagram to show how a small gall-stone may obstruct the papilla, and, if the ampulla of Vater be very large, may convert the common bile-duct and duct of Wirsung into one canal, thus predisposing to acute pancreatitis. *b*, Diagram to show a method of termination of the ducts which will not predispose to pancreatitis (Opie).

From the above observations it is evident that gall-stones in the common duct and in the ampulla are the most frequent causes of the various forms of pancreatitis.

In a certain percentage of cases the bile-duct and duct of Wirsung open by separate orifices into the duodenum (Fig. 367), while in others the duct of Santorini is the chief outlet of the pancreas or is of sufficient size to take on the functions of the duct of Wirsung, if the latter becomes obstructed.

In the case of injury (traumatism) to the pancreas, it is believed that the secretion of the gland may act on the walls of the blood-vessels and produce further hemorrhage, and infection from the duodenum may be an additional factor in causing an acute inflammation.

Hemorrhage into the pancreas may also arise from diseased blood-vessels, and the injection of mercury in a syphilitic subject has been reported as producing acute pancreatitis, resulting possibly from increased secretion and congestion of the gland. Syphilis alone, however, may be a factor.

Infection of the biliary passages by the typhoid bacilli is not an infrequent occurrence, and in one case they were proved to be the factor in pancreatic catarrh. Probably influenza and some of the other infectious diseases may cause pancreatitis.

Pancreatitis has been reported in a number of cases occurring as a complication of mumps. Out of 652 cases treated at the military hospital of Val de Grâce, Simonin reports 10 cases, or 1.3 per cent., in which symptoms of pancreatitis occurred from the first to the twelfth days of the disease, and which lasted from two to seven days. The chief symptoms were epigastric pain and tenderness associated with nausea and vomiting. Jacob reports a case in which a tender swelling was found in the epigastric region (an enlarged pancreas); and Lemoine and Lapasset describe a case occurring on the fifteenth day in which autopsy showed an acute inflammation of the pancreas. Epigastric pain, jaundice, and hematemesis were the chief symptoms.

With pyemia, one has the general symptoms of the disease with the additional epigastric pain, tenderness, etc., when the pancreas is involved. Syphilis may affect the pancreas as a tertiary lesion (gumma) or it may be congenital. It causes an interlobular interstitial pancreatitis. Ulceration of the stomach or duodenum may spread to the pancreas and may produce suppuration in that organ. The relation of alcohol to the production of cirrhosis of the pancreas has been questioned. It has been found in association with cirrhosis of the liver, and the writer sees no particular reason why this factor should not be identical. Of course, abuse of alcohol may produce a gastro-intestinal catarrh, which, by extension, might be the cause of the chronic pancreatitis. Arteriosclerosis, the result of alcohol, might again be the cause of pancreatic involvement.

Cirrhosis of the pancreas, usually resulting in a fatal diabetes, in some cases undoubtedly results from a catarrh of the ducts of long duration, which first produces an interlobular and later an interacinar pancreatitis. Unquestionably, arteriosclerosis may be a cause of chronic pancreatitis.

## CATARRH OF THE PANCREAS

**Acute Catarrh of the Pancreas.**—This type of pancreatitis resembles infective cholangitis, with which it is usually associated, and is generally due to a stone in the common bile-duct or diverticulum of Vater, with incomplete obstruction. The process is undoubtedly one of extension into the pancreatic duct. The writer *does not believe* that infective cholangitis and *acute catarrhal pancreatitis* can be *differentiated,* and thinks that the latter is a sequel to the former. The symptoms of infective cholangitis are: Variations in the intensity of the jaundice, which is of long duration, usually over a year; the liver may be of normal size or slightly enlarged; the gall-bladder is not *distended;* the spleen is enlarged; there is no ascites; bile may be present in the feces continuously or occasionally; and at times there may be ague-like paroxysms, with chilly fever and sweating (the hepatic intermittent fever of Charcot). On these occasions there may be colicky pains and the jaundice deepens, and often nausea and vomiting are present. The chills are often quite severe and the temperature may rise to 103° to 105° F. Between attacks the temperature is normal. Though this condition may continue for years without suppuration within the ducts, the process is one of infection, and operative procedure is indicated, *i. e.*, the removal of the impacted stone. The infective cholangitis and acute catarrh of the pancreas are together cured by this method.

**Chronic Catarrh of the Pancreas.**—The possibility of diagnosing chronic catarrh of the pancreas is at present a matter of much dispute. Curtin[1] believes that there is little evidence that it is an inflammatory process. Lando[2] holds that stagnation of the pancreatic secretion within the acini may produce chronic interstitial pancreatitis.

Robson[3] and Cammidge believe that chronic catarrh of the pancreas can usually be diagnosed by digestive and metabolic symptoms and by swelling of the gland, and that the symptoms and physical signs resemble in a mild degree those of chronic pancreatitis.

*Etiology.*—The most frequent cause of chronic catarrh of the pancreas the writer believes to be a gastroduodenitis or duodenitis. These conditions may be either acute or chronic, and as a result an infection of low grade may occur, both of the common bile-duct and of the duct of Wirsung. While a catarrhal condition of the duodenum exists, as can be determined by the microscopic appearance of mucus from the small intestine, "testing the intestinal functions" will be of value chiefly to show the catarrh. This last will interfere with the secretion of the prosecretin and enterokinase, and, even if the pancreatic duct were not involved, or to a slight extent, and the common bile-duct were chiefly affected, the functions of the pancreas would be interfered with. After the *mucus has disappeared from the stools* and jaundice still persists, if *then one finds the pancreatic functions markedly disturbed*, the assumption of catarrh of the pancreatic duct is a fair one. Moreover, persistent jaundice following the history of a gastro-intestinal or intestinal catarrh shows that the common bile-duct is involved, and *probably the pancreatic duct.*

*Treatment.*—The general medical treatment is that of duodenal jaundice (see Intestinal Catarrh). This may be continued for the chronic condition, and if failure results, cholecystotomy or cholecystenterostomy may be performed, which will relieve the catarrh of the common bile-duct and take off pressure from the pancreatic duct.

The other most common cause of chronic pancreatic catarrh is an impacted stone in the pancreatic portion of the common bile-duct, which presses on the pancreatic duct, or a stone in the ampulla.

The obstruction is usually partial, and the grade of infection producing a chronic catarrh is low.

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[1] Phila. Hosp. Rep., 1902, v.
[2] Zeit. f. Heilk., 1906, Hft. 1.
[3] The Pancreas: Its Surgery and Pathology.

The *history is one of gall-stone attack, followed by jaundice.* In some cases the pancreatic functions may be disturbed. One must remember that in some the duct of Santorini may take on in part the function of the inflamed duct of Wirsung, so that the examination of the pancreatic functions may not afford information. Furthermore, the repeated passage of gall-stones through the common duct may set up a catarrhal condition which may extend to the pancreatic ducts.

Regarding the determination of pancreatic enlargement in chronic catarrh of the pancreas the author is very skeptical.

The removal of the impacted stone affords relief both to the catarrh of the common bile-duct and to that of the pancreatic duct.

In a large percentage of cases the absolute determination and early diagnosis of chronic catarrh of the pancreatic duct is an impossibility. Its probability can be reasoned out, and the treatment is fortunately the same as that of chronic catarrh of the common bile-duct.

**Suppurative Catarrh of the Pancreas.**—This disease bears the same relation to catarrh of the pancreas as does suppurative cholangitis to simple catarrhal jaundice. Suppurative cholangitis and suppurative catarrh of the pancreas are usually associated.

*Etiology.*—The usual cause of this disease are gall-stones. One can explain that the same cause may produce in one case a simple catarrh and in another suppuration, on the ground that in suppurative cases there must be a high grade of infection with a lowered resistance on the part of the patient.

Abscesses of the liver, gall-bladder, and pancreas are usually present.

*Symptoms.*—In the milder cases the symptoms are septicemic, in some those of a local abscess of the pancreas, and in the more severe cases they are pyemic.

The preliminary history is one of attacks of gall-stones with subsequent jaundice, enlarged liver, tenderness and pain in the epigastrium and right hypochondrium, muscular rigidity, frequently a mass in these regions, irregular septic temperature, chills, sweating, leukocytosis, and increased polynuclears. Cammidge found his urine reaction in one of these cases of septicemic type.

Occasionally, suppurative catarrh of the pancreas may assume a subacute form and end in a single pancreatic abscess with the symptoms associated therewith. Even after drainage of a simple abscess of the pancreas, interstitial pancreatitis may occur and ultimately lead to the death of the patient.

## CHAPTER XL

# ACUTE PANCREATITIS—CHRONIC PANCREATITIS

## ACUTE PANCREATITIS

THE best classification of acute inflammation of the pancreas has been made by Fitz:[1]

1. Hemorrhagic pancreatitis, or hemorrhagic necrosis of the pancreas (Opie).
2. Gangrenous pancreatitis.
3. Suppurative pancreatitis.

Gangrenous pancreatitis, though due to other conditions, is usually the result of hemorrhagic pancreatitis, and in about 50 per cent. of cases recorded there is evidence of previous hemorrhage in the gland. The symptoms of the two conditions resemble each other, but with gangrenous inflammation the disease is of longer duration. *Disseminated fat necrosis* is present with both the *hemorrhagic* and *gangrenous types*. Suppurative pancreatitis results from hemorrhagic necrosis of the pancreas, necrotic tissue being particularly susceptible to bacterial infection. Pancreatic abscess also occurs in association with cholelithiasis through ascending infection from the intestinal tract, or from suppurative cholangitis, carcinoma, compressing the pancreatic duct, pancreatic calculi, perforating ulcer in an adjacent organ, and traumatism.

*Fat necrosis is uncommon with suppurative inflammation of the pancreas.*

It is interesting to note that an acute interstitial inflammation of the pancreas without suppuration (acute interstitial pancreatitis) may occasionally occur.

### ACUTE HEMORRHAGIC PANCREATITIS

(*Synonym.*—Hemorrhagic Necrosis of the Pancreas—Opie.)

This lesion has really not the characteristics of an inflammatory process. There is a primary necrosis of the parenchyma of the pancreas, and the only inflammatory changes which occur are at the margin of the necrotic tissue. It should properly be called "hemorrhagic necrosis of the pancreas." Fat necrosis which accompanies it has been demonstrated as due to the fat-splitting enzyme of the pancreatic juice.

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[1] Acute Pancreatitis, Med. Rec., xxxv, 197, 225, 253, 1889.

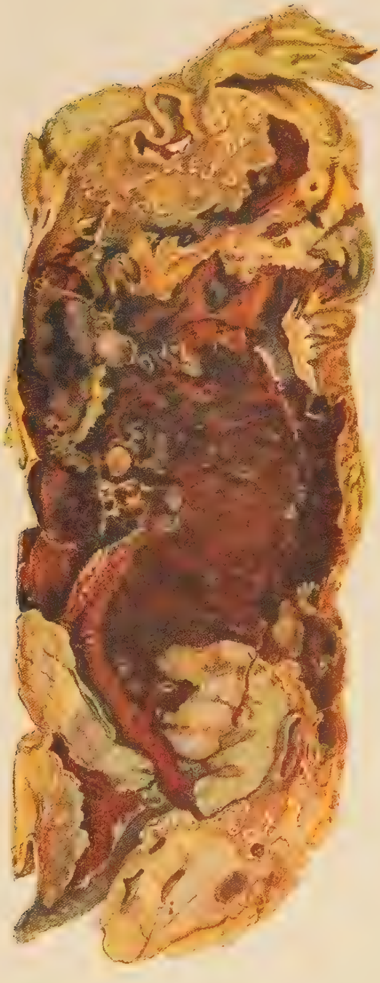

Fig. 368.—Pancreas and adjacent tissues from a case of acute hemorrhagic pancreatitis with fat necrosis (St. Bartholomew's Hospital Museum).

**Pancreatic Hemorrhage.**—Hemorrhage into the substance of the pancreas may be due to a variety of causes. It may accompany tumors or may occur in pancreatic cysts, or with purpura, eclampsia, and the acute infectious diseases. Such cases have nothing in common with hemorrhagic pancreatic necrosis.

**Experimental Hemorrhagic Necrosis of the Pancreas.**—Hemorrhagic pancreatitis has been experimentally produced on dogs by the injection of various substances into the duct of Wirsung, such as the deliquescent chlorid of zinc, artificial gastric juice, papain, the diphtheria toxin, hydrochloric acid, nitric and chromic acid,[1] suspensions of bacteria, etc. Bile[2] injected into the pancreatic duct almost uniformly produced the same results.

Flexner[3] demonstrated that the *production of the lesion* may be attributed to *the bile salts.*

**Occurrence.**—Hemorrhagic necrosis of the pancreas is found more frequently in men than in women. Peiser[4] tabulated 121 cases of hemorrhagic and gangrenous pancreatitis, of which 79 occurred in men and 42 in women.

Körte[5] collected 37 cases of the hemorrhagic type in males and 4 in females. In cases with gangrenous pancreatitis, 21 were in males and 19 in females. Individuals who are apparently in good health are not infrequently attacked, and fat patients are believed to be *particularly susceptible.*

**Age.**—Hemorrhagic necrosis of the pancreas and gangrenous pancreatitis occur usually between twenty and sixty years of age.

**Etiology.**—*Bacterial Infection.*—Opie[6] holds that no relation between hemorrhagic necrosis of the pancreas and bacterial invasion has been demonstrated. Welch[7] believes that the organisms penetrate the tissue subsequent to the production of the lesion.

Various investigators have found the colon bacillus, the pneumococcus, streptococci, staphylococci, and other bacteria in acute hemorrhagic pancreatitis. Most agree that, as the findings are inconstant and as the necrotic parenchyma may contain no micro-organisms, they are secondary invaders of the injured tissue. In view of gall-bladder infection produced by the colon bacillus, the author believes that in some instances the colon bacillus may, in part, be responsible for this type of acute pancreatitis. It is further interesting to note that in an autopsy of one of Robson's[8] cases, performed three hours after death, subsequent to operation for acute hemorrhagic pancreatitis, blood-cultures contained the bacillus coli communis, as did also the pancreas.

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[1] Contributions to the Science of Medicine, Johns Hopkins Hosp. Rep., 1900, ix, 793.

[2] Opie, Bull. of Johns Hopkins Hosp., 1901, xii, 182.

[3] Jour. of Exper. Med., 1906, viii, 167.

[4] Deutsche Zeit. f. Chir., 1902, lxv, 302.

[5] Chirurg. Krankheit, des Pancreas, Deutsche Chirurg., Stuttgart, 1898.

[6] Disease of the Pancreas.

[7] Med. News, 1891, lix, 669.

[8] The Pancreas (Robson and Cammidge), p. 392.

**Cholelithiasis.**—There is a close relationship between cholelithiasis and acute pancreatitis. Egdahl has found that out of 105 cases of acute pancreatitis, cholelithiasis was present in 42 per cent. This is probably a minimum percentage. The calculus is sufficiently large to occlude the opening of the ampulla of Vater into the duodenum, and yet so small that it will not fill up the ampulla and will not obstruct the orifices of the common bile-duct and of the duct of Wirsung. These two ducts are converted into a continuous closed channel, and the bile entering the pancreas through its duct produces a hemorrhagic necrosis of the gland. In only a little more than three out of ten persons is the size of the diverticulum of Vater such that the stone will block the opening into the duodenum and yet not obstruct the duct openings. In 1 out of 10 cases the bile-duct joins the smaller pancreatic duct, and, furthermore, in some cases the duct of Santorini is the chief duct of the pancreas. In other cases the common duct and duct of Wirsung enter the bowel separately. These facts explain the comparative rarity of hemorrhagic necrosis of the pancreas when compared with the frequency of cholelithiasis.

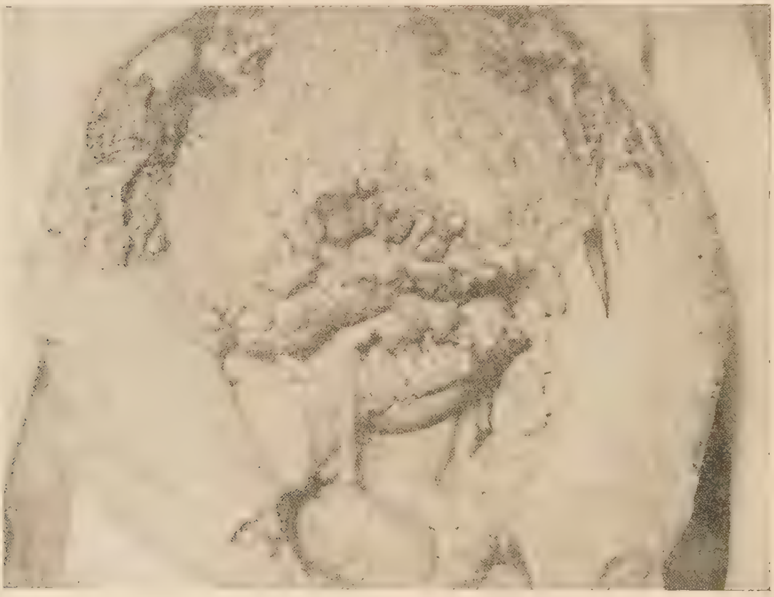

Fig. 369.—Areas of fat necrosis in the mesenteric and omental fat and in the abdominal wall in a case of acute hemorrhagic pancreatitis (Fison).

Opie reports 8 cases of hemorrhagic necrosis of the pancreas, in which gall-stones were present in 5.

*Penetration of the Duodenal Contents into the Pancreatic Ducts.*—In about one out of ten persons the duct of Santorini is the *larger*, and is the chief outlet of the gland. Probably a duct of this type can be the port of entry of the duodenal contents.

*Traumatic Necrosis.*—A blow, an injury to the abdomen in the epigastric region, or a stab-wound may produce hemorrhage into the pancreas. Crushing of the gland, associated with occlusion of the vessels (venous thrombosis), together with an escape of the pancreatic juice into the damaged area, may be subsequently followed by hemorrhagic necrosis.

Alcoholism and diabetes have by some been considered factors in the production of this type of acute pancreatitis.

Parturition has been mentioned as a cause, the pancreas being affected in a similar way as the kidneys and liver by the toxemias of pregnancy. The theory has been advanced that the pancreatic necrosis may result from embolism of the giant cells of the placenta.

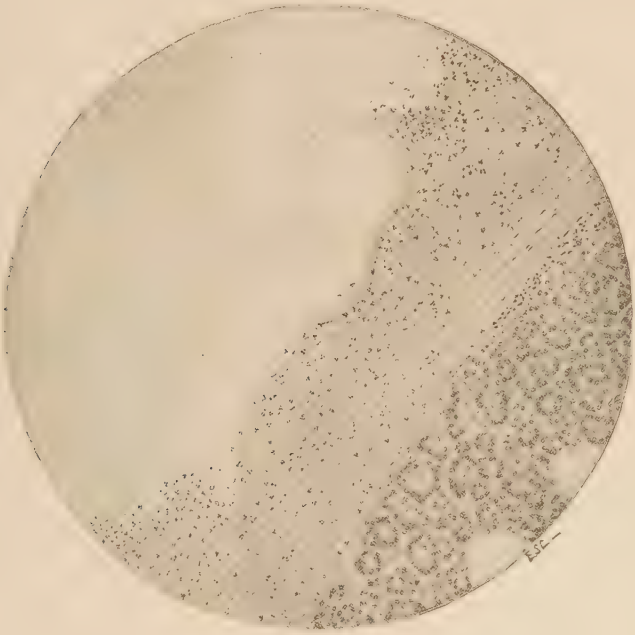

Fig. 370.—Hemorrhagic necrosis of the pancreas, showing abrupt transition from normal to necrotic tissue. At the margin of the living tissue are red blood-corpuscles, leukocytes, and fibrin (Opie, "Diseases of the Pancreas").

Undoubtedly the *major* number of cases of hemorrhagic necrosis of the pancreas *can be imputed to a gall-stone impacted in the mouth of the ampulla of Vater.*

**Pathology.**—The pancreas is enlarged, firm, and generally covered by clotted, black-red blood (Fig. 368). There is blood-stained fluid in the lesser peritoneal cavity, and frequently blood infiltrates the tissues about the pancreas. Fat necrosis always accompanies it (Fig. 369). Usually the entire organ is not affected, and there may be areas of normal parenchyma.

There is coagulation necrosis in the diseased areas, involving the epithelial cells, interstitial tissue, and the blood-vessels. There is an abrupt transition from the necrosed area to living tissue, and there is a line of demarcation, a narrow zone, which contains fragments of nuclei, red blood-cells, polynuclear leukocytes, and fibrin (Fig. 370).

Acute inflammation is not always present. There is hyaline thrombosis of the capillaries within the tissue which is in contact with the necrotic area. In some cases hemorrhage may be a slight feature and necrosis the chief lesion, with a few hemorrhagic areas.

Complete recovery may occur, and the necrotic area become absorbed and replaced by fibrous tissue.

After healing of the pancreatic lesion, the lesser peritoneal cavity may still contain opaque fluid and necrotic particles, and the pancreas be covered by black, altered blood.

As gangrenous pancreatitis is a progressive stage of the acute hemorrhagic type, it will be next described, and the symptoms of both conditions will be given later.

## GANGRENOUS PANCREATITIS

**Stage of Gangrene.**—*Etiology.*—This condition is *usually a sequel of hemorrhagic necrosis of the pancreas* should the patient survive a sufficient period of time. Perforating ulcer of the stomach, intestines, or biliary tract, or even extension of a cholangitis through the pancreatic duct, may be causes. Traumatism may first produce a hemorrhagic necrosis of the pancreas with subsequent gangrene.

**Pathology.**—The pancreas is usually swollen, dark red, or red-gray, or slate colored, generally foul smelling, and is often surrounded by a thin greenish or dark-colored purulent fluid. It may lie free in the cavity of the omentum, which last is distended with ill-smelling bloody or blackish fluid.

In some cases in about a week or ten days the pancreas may be dry, firm, and of dark-brown color, covered by altered blood. In the parenchyma of the gland yellow spots of softening may alternate with areas of hemorrhage.

In others, in about two weeks the pancreas may appear a black, soft, friable mass, and the cavity of the lesser omentum contain chocolate-colored fluid with blackish clots.

The gangrenous condition of the gland is produced by the invasion of bacteria, which produce these changes in a necrotic gland containing extravasation of blood.

Suppuration with or without perforation of the gastro-intestinal tract is a secondary condition. The microscopic appearance of gangrene is quite typic. *Disseminated fat necrosis is present.*

*Accumulation of Fluid in the Lesser Peritoneal Cavity.*—With these types of acute pancreatitis there is accumulation of fluid in the lesser peritoneal cavity. The foramen of Winslow is closed by adhesions. The fluid usually contains the products of pancreatic secretion. The contents may at first be sterile, but later become infected from the gangrenous pancreas. The bacillus coli, bacillus lactis aërogenes, bacillus proteus vulgaris, and streptococcus pyogenes have been found in this sac.

An abscess may form, the wall of which is blackish or gray, and consists chiefly of necrotic fat. The contained fluid may be brown or gray in color, and contain soft, greasy, necrotic material. Through erosion the abdominal wall in the left lumbar region may be perforated, or perforation into the general peritoneal cavity may occur. A subdiaphragmatic abscess may form, and the diaphragm may be perforated with a resulting empyema. Perforation of the stomach, duodenum, or transverse colon may take place, and the discharge of necrotic pancreatic tissue from the bowel has been recorded.

### SYMPTOMS OF ACUTE PANCREATITIS (HEMORRHAGIC AND GANGRENOUS)

**Symptoms of Hemorrhagic Necrosis.**—The patient may have been previously healthy or a sufferer from occasional attacks of indigestion. In some there may be a history of gall-stone attack. Suddenly there is an intense pain in the upper abdomen, the epigastric region, followed by vomiting, more or less obstinate in character, and severe collapse, which has in a few hours even caused death in some cases reported. The pain, which is very intense, never wholly subsides, and is paroxysmal and increases on movement. If the condition persists for a longer period, in the course of twenty-four hours there is an epigastric swelling, tympanitic or resistant, with tenderness on pressure, which is present from above the umbilicus to the ensiform process and over the pancreas. Obstinate constipation occurs, though the obstruction is not absolute, as flatus occasionally passes and a large enema may secure a movement. A normal or, usually, subnormal temperature is present in the very acute cases. If the case survives several days there may be irregular temperature, or occasionally it may be high. The pulse is rapid and small and cyanosis may result, both of the face and abdominal wall particularly (Halsted). The aspect is anxious and the face pinched. An *epigastric peritonitis* is present at first, which later may become general, and general distention occur. Tender spots are also often present throughout the abdomen. They are believed to lie over the areas of fat necrosis. Jaundice may be present, at first slight, which may later deepen. The vomitus may consist, first, of food; later, of bile, and, finally, of black altered blood. Hemorrhage may also occur from the other mucous surfaces, the intestines, subcutaneously, and into the skin. Death usually occurs on the second to fifth day, though a fatal issue may take place within a few hours. Delirium may occur before death. This condition may be mistaken for intestinal obstruction, but stercoraceous vomiting and visible peristalsis of the intestines are absent, which occur in the latter. On laparotomy, areas of fat necrosis are visible, which are at once suggestive of the condition.

**Subacute Pancreatitis.**—The writer has recently seen a case in which there was an acute attack of pain, apparently from a gall-stone; tenderness over the pancreas and common duct; vomiting of blood and passage of blood in the stools; no jaundice and no shock of marked degree. Recovery ensued. The stone evidently escaped into the gut.

**Symptoms of Gangrene of the Pancreas.**—With this condition the violent symptoms of an acute hemorrhagic pancreatitis, which may precede it, may diminish in their severity, or the symptoms of the onset may be less severe in character. The patient survives the more acute attack and the condition is more chronic. Opportunity is given for bacterial invasion and subsequent gangrene. The pain is localized in the epigastric region and vomiting may occur at intervals. Symptoms pointing to suppuration eventually give evidence that infection has occurred. There are frequently an irregular temperature and chills. Diarrhea may later be present. An ill-defined tumor may at times be felt above the umbilicus, due to accumulation of fluid in the lesser peritoneal cavity, or to hemorrhagic or purulent fluid about the pancreas. The mass is generally situated in the epigastrium, and extends toward the spleen. Inflation of the stomach and colon may aid in locating its position. The stomach separates the tumor from the liver, and, when inflated, covers it. At times erosion of tissue over the left kidney occurs, and there may be a swelling below the left costal margin as far as the iliac crest.

The gangrenous type of pancreatitis may be prolonged for several weeks or longer. Considerable loss of weight may occur in these cases when prolonged.

In both these types of acute pancreatitis, digestive disturbances, such as have been described under the General Symptoms of Diseases of the Pancreas, may occur. Seldom is there such destruction of the pancreas that its functional activity is entirely destroyed. Sugar is rarely present; fatty stools are rare.

**Leukocytosis.**—Leukocytosis may be present during the first few days of the attack of hemorrhagic necrosis of the pancreas. It is not always present, however. With gangrenous pancreatitis, leukocytosis may vary from 15,000 to 40,000. It may diminish, and there may even be leukopenia before death. Increased polynuclears occur with leukocytosis. Blood clotting is delayed.

**Diagnosis and Differential Diagnosis.**—The anamnesis, the symptoms of acute epigastric peritonitis, pain, vomiting and collapse, and the fulminating character of the attack are at once suggestive. The writer believes that determination of the pancreatic functions, the Cammidge reaction, etc., are of no practical assistance. The condition is acute, and radical procedure is indicated, *with no delay.*

Laparotomy reveals fat necrosis, which is diagnostic in connection with the symptoms.

With intestinal obstruction there are stercoraceous vomiting and intestinal paralysis; no flatus is passed. With perforation of duodenal or gastric ulcer the history, gravitation of the contents toward the pelvis, and the absence of liver dulness are serviceable data.

With phlegmonous cholecystitis the swelling and tenderness usually first lie below the right costal margin. Determination of the gall-bladder zone (Head) is of service.

With appendicitis epigastric pain may first be present, but tenderness at McBurney's point and muscular rigidity over the appendix are present. Head's zone for the appendix is also diagnostic.

**Treatment.**—Collapse must receive treatment at once and the acute pain must be relieved, which is in itself a serious factor in producing the shock. Morphin, $\frac{1}{4}$ to $\frac{1}{2}$ grain (0.015–0.03), may be given by hypodermic injection, and strychnin, $\frac{1}{30}$ grain (0.002), camphor oil, 5 grains (0.3) camphor in 15 minims (1.0) of sterile olive oil or almond oil, should be administered. Hypodermoclysis may be indicated. Vomiting should be relieved by lavage. Hot saline enemata, 1 pint to 1 quart (500–1000 c.c.) each, at 120° F., may be given for shock. A nutritive enema may be added, and in the latter 20 grains of lactate of calcium should be given to overcome the hemorrhagic tendency. Chlorid or, preferably, lactate of calcium, 60 to 90 grains (1–1$\frac{1}{2}$ drams) daily, should be given in divided doses, 15 grains (1.0) at a time, by enema, both before and after operation, in the latter event for ten days. In a recent case with hematemesis and inability to retain the enema, 10 grains (0.6) of lactate of calcium were dissolved in 5 ounces of water and 1-dram doses of this solution given every one-half to one-quarter hour with success. This was repeated several times daily. Human blood-serum may also be injected hypodermically, if necessary, to combat the hemorrhagic tendency, or horse serum by mouth. Continuous enteroclysis with hot saline at 120° F. will both relieve distention and stimulate the patient. Recourse to laparotomy is indicated as soon as the shock is relieved. Incision is made in the median line or over the most prominent part of the tumor, if such can be determined. On opening the abdomen the presence of fat necrosis is at once diagnostic of pancreatic involvement.

The cavity of the lesser peritoneum is then entered through the gastrocolic ligament, and fluid and necrotic material, if present, are evacuated. The pancreas should also be drained. The operator should determine the condition of the bile-passages, and if the shock of the operation be not dangerously increased by further procedure, gall-stones, if present, should be removed from the gall-bladder and any impacted stone from the common duct. If prolongation of the operation seem inadvisable, the gall-bladder should be drained, and by a subsequent operation the stones removed therefrom and the impacted stone cleared from the ampulla. Proctoclysis subsequent to operation is of value for the sepsis.

### SUPPURATIVE PANCREATITIS

**Pathology.**—The pancreas is enlarged. There may be a diffuse suppurative process of the gland or multiple small abscesses, in which the process is more acute, or there may be a single abscess of the pancreas (subacute pancreatitis—Robson), a less acute process, and which may even be of considerable duration. Rarely, there may be an acute interstitial pancreatitis without suppuration—an intermediary stage.

**Etiology.**—Suppurative pancreatitis may be secondary to hemorrhagic necrosis of the pancreas, or quite frequently result from ascending infection of the pancreatic duct. It may occur in association with cholelithiasis by occlusion of the duct of Wirsung by a calculus lodged in the diverticulum of Vater. Suppurative cholangitis or cyst of the pancreas may be followed by suppuration.

Pancreatic calculi, carcinoma compressing the pancreatic duct, carcinoma of the diverticulum of Vater, or ulceration of neighboring organs may produce abscess of the pancreas. Traumatism and digestive disturbances have been considered factors.

*Bacterial Infection.*—Among the micro-organisms isolated from abscess of the pancreas are Bacillus coli, pyogenic cocci, proteus vulgaris, and diplococcus lanceolatus. Colon bacillus infection the author believes to be a possible etiologic factor.

*Fat necrosis rarely accompanies suppurative pancreatitis.*

**Sequelæ of Suppurative Pancreatitis.**—Infection of the lesser peritoneal cavity is frequent. Perforation into the general peritoneum may result. The abscess has ruptured into the stomach, with resulting vomiting of pus, or has perforated the bowel, with discharge of pus therefrom. Thrombosis, with infection of the splenic and portal veins and metastatic abscess of the liver, may occur. The pus may burrow into the right loin and be mistaken for a perirenal abscess. It may find its way into the left iliac region or form a subphrenic abscess. It may burrow between the liver and stomach, or reach the surface above or below the latter, or pass into either loin or into the pelvis, or into the left broad ligament.

Even with proper drainage, chronic interstitial pancreatitis may result, from which the patient may succumb.

**Symptoms of Suppurative Pancreatitis.**—If this condition results from hemorrhagic necrosis of the pancreas, cholelithiasis, pancreatic cyst, or pancreatic lithiasis, its symptoms are modified by the preceding condition. In about 50 per cent. of the cases there are first a sudden onset, with intense epigastric pain, vomiting and collapse, the symptoms of hemorrhagic pancreatitis, of which suppurative pancreatitis may be a sequel. The severity of the symptoms then lessens and the course becomes chronic. In others there may be a more or less sudden onset with severe pain, vomiting, and constipation, but collapse is not a marked symptom and, as a rule, is absent.

The upper region of the abdomen in these cases does not become as rapidly distended, and vomiting is less severe. In others the onset is gradual and the symptoms are less severe. The abdominal pain may be little more than discomfort and gastric disturbances occur at times. In some cases a history of intermittent attacks of pain, at first without and later with jaundice, may be elicited, due to gall-stones, and in others, symptoms of infective cholangitis, with rigors, intermittent fever, and deepening of the jaundice.

Thayer[1] reports a case of jaundice due to pressure of a pancreatic abscess on the common bile-duct in which carcinoma was suspected.

Tenderness over the pancreas is usually well marked, and in about one-fourth of the cases a palpable tumor can be determined. This is more frequently due to accumulation of inflammatory products within the lesser peritoneal cavity than to a palpable pancreas.

Constipation may be followed by diarrhea, which may be fetid in character if pus or blood are present in the stools. Fatty stools may occasionally be observed or impaired absorption of fat may at times be determined. Robson believes that fat and undigested muscle-fibers are usually present in the stools, and Opie, that they rarely occur. Glycosuria is rare.

The writer believes that *fat in the stools and glycosuria are rare occurrences*. In the subacute or chronic type of case the pulse is not seriously affected. In many cases there is fever, reaching up to 105° F., with recurring chills. The morning temperature may be normal. Leukocytosis is present and increased polynuclears. There is a gradual loss of flesh and the patient becomes more feeble. Albuminuria is quite frequently present. Robson holds that the Cammidge reaction is, as a rule, well marked. The author believes it may be tried, but doubts its diagnostic value.

Suppurative pancreatitis with single abscess formation tends to take a chronic course, from one to even eleven months. With diffuse suppuration, liver abscess may be associated and the symptoms may be pyemic.

There may be evidence of abscess formation in the regions described under Sequelæ of Suppurative Pancreatitis.

Rarely, there is no elevation of temperature, and when, in addition, the mass cannot be palpated, pancreatic abscess cannot be determined.

**Diagnosis.**—Early occurrence of fever, with chills, epigastric tenderness, pain and swelling, and leukocytosis, suggests pancreatic abscess. When it follows hemorrhagic necrosis of the pancreas the diagnosis is more difficult, since when this condition reaches the stage of gangrene, abscess of the lesser peritoneum may also be present, as it is also in suppurative pancreatitis. A chronic course, however, suggests suppuration. *Fat necrosis is also usually absent.*

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[1] Amer. Med., 1902, 341.

**Treatment.**—During the acute stage the treatment of vomiting, collapse, and distention are the same as with the other types of acute pancreatitis already described.

Enemata of soapsuds, with 1 dram of spirits of turpentine, or enteroclysis aid in relieving distention. Calomel is of value to empty the bowel during the constipated stage, and can be given in a dose of 5 grains (0.3), or it may be administered in small doses as an intestinal antiseptic, in either case to be followed by a saline cathartic. Lactate of calcium, 60 grains (4.0), should be given daily in divided doses before and after operation to prevent hemorrhage, or if such is occurring. The urine should be examined for colon bacilli and urotropin and benzoate of soda, 80 grains (5.3) each, should be given by enema in divided doses daily if such are present.

Early operation with drainage of the abscess is indicated; also the removal of biliary or pancreatic calculi if such are present.

## CHRONIC PANCREATITIS

In undertaking to give the reader a clear and concise conception of chronic interstitial pancreatitis, so that he may readily be enabled to diagnose this condition, the writer, in the present state of our medical knowledge, is confronted by an almost impossible task. Though the study of this lesion or, rather, of one particular type of it, has contributed important facts to our knowledge of diabetes mellitus, yet chronic pancreatitis of another anatomic type, at times difficult to diagnose as a result of various factors, may occur. The symptoms appearing with the primary disease, such as inflammatory conditions of the gall-bladder, duodenum, etc., which may stand in a direct causative relation to the chronic pancreatitis, may greatly obscure the diagnosis. One must frequently arrive at the latter by the process of exclusion. Chronic inflammation of the pancreas may be present, and, on account of anatomic peculiarities of the gland found in a percentage of cases, the disease may become well advanced before disturbances of the pancreatic functions can be determined. Opie, in fact, holds the view that " the lesion is seldom associated with such definite symptoms as to be recognized during life, and that, even at autopsy, the condition is frequently overlooked."

Robson and Cammidge, on the contrary, believe that " from examination of the patient, the history of the case, and the results of chemic and microscopic examination of the excreta, a correct opinion may be formed in a large majority of instances." These represent extreme views. The writer believes that in some cases the diagnosis of chronic pancreatitis is possible, while in other instances, especially in the early cases, our present medical knowledge is not sufficiently advanced to permit accurate determination of this lesion.

**Sex.**—In 30 of Opie's[1] cases, 17 occurred in males and 13 in females; while in Deaver's[2] patients, 22 were males and 16 females. Böhm reports 65 per cent. males and 35 per cent. females. The disease, therefore, preponderates in the male sex.

The relation of sex to chronic pancreatitis, as compared with cholelithiasis, is reversed, biliary affections preponderating in the female.

**Age.**—Opie has collected 30 cases from the age of ten to eighty years. Twenty, or two-thirds, occurred between forty and sixty years. These included postmortem records. Deaver reports 34 cases, in which 4 were under thirty years of age and the balance between thirty and sixty years. Two-thirds of these cases occurred between forty and sixty years. All but one were operative.

**Etiology.**—In about one-half to two-thirds of all cases of chronic pancreatitis, cholecystitis and cholelithiasis are found to be the exciting causes.

Deaver found disease of the gall-bladder or ducts in 65 per cent., and one-half had gall-stones at operation; Mayo found gall-stones in 81 per cent.; Robson, in 60 per cent.

A *large calculus* in the diverticulum of Vater or in the common duct above its junction with the pancreatic duct may compress the latter and cause chronic inflammation of the pancreas. Bacterial infection (ascending infection) of the duct of Wirsung is probably a factor. A small calculus in the orifice of the diverticulum of Vater usually produces acute pancreatitis. Flexner reports from his experiments that when the composition of the bile is modified by a diminution of its salts or by an increase of colloid material, its entrance into the pancreatic duct is likely to set up a chronic pancreatitis, while fresh unaltered bile sets up acute changes. Small stones sometimes cause chronic pancreatitis, so that modified bile in these cases has probably been diverted into the duct.

**Malignant Growths.**—A growth compressing or invading the pancreas may produce chronic pancreatitis: it may either compress the duct of Wirsung or obstruct the ampulla or papilla, interfering with the flow of pancreatic secretion. Infection from an ulcerated surface may be a factor. The pancreas may be invaded by a carcinoma of the stomach, with resulting local or diffuse interstitial inflammation.

*Impacted pancreatic calculus, stenosis of the duodenal orifice of the gland* following ulceration, and hydatid membrane obstructing the opening are reported as causes.

*Ascending Infection from the Duodenum.*—Chronic pancreatitis may result from extension of a duodenal catarrh into the pancreatic duct. Associated bacterial infection is a factor even when the ducts are blocked by calculi.

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[1] Disease of the Pancreas.

[2] Jour. Amer. Med. Assoc., April 15, 1911; ibid., July 1, 1911.

In the latter event, bacteria may enter along the walls of the inflamed duct through the lymphatics or blood-stream.

*Infectious Diseases and from Adjacent Organs.*—Chronic pancreatitis may occasionally occur as a sequel to typhoid fever, mumps, influenza, and other zymotic diseases. Extension of the inflammatory process from an adjacent organ, as in a case of gastric ulcer or carcinoma of the pylorus, may produce it. Among other causes are arteriosclerosis and *chronic passive congestion of the pancreas,* due to chronic diseases of the heart, lungs, and liver.

The action of toxic substances in the blood may produce a chronic pancreatitis, notably tuberculosis, syphilis, and alcohol.

Chronic pancreatitis is quite frequently associated with cirrhosis of the liver. In 30 of Opie's cases of chronic pancreatitis, cirrhosis of the liver was present in 8.

Lefas[1] and Opie[2] find that chronic pancreatitis may accompany either the atrophic form of cirrhosis of the liver (Laennec's) or the hypertrophic form. In the former case the type of pancreatitis is intralobular (interacinar), and the weight of that organ is increased, while in the latter case the interlobular type occurs, and there is no increase in the volume of the pancreas. Cirrhosis of the liver and interacinar pancreatitis are associated with diabetes mellitus with hemachromatosis.

The writer has recently reported a case of undoubted chronic pancreatitis with marked colon bacilluria. *He believes the colon bacillus responsible in some cases.* This patient had steatorrhea.

**Pathology.**—There are two types of chronic interstitial inflammation of the pancreas—the *interlobular* and the *interacinar.*

*Chronic Interlobular Pancreatitis.—Etiology.*—This type is most frequently due to obstruction of the pancreatic duct or to ascending infection along the duct.

The interlobular tissue is the site of the chief change, and the gland is dense, hard, and the surface is nodular or granular. The tissue on section is compact and homogeneous, the areolar tissue being replaced by scar-like bands. Occasionally the gross appearance may be little altered, so that the lesion can only be recognized under the microscope. Considerable fat may infiltrate the newly formed tissue, and small foci of fat necrosis may be present.

Coarse bands of fibrous tissue separate the lobules of the parenchyma, and lymphoid cells, plasma cells, and eosinophiles are present in the interstitial tissue. The chief increase of fibrous tissue is between the lobules. Acini, with atrophied nuclei and dilated laminæ, are at times separated by new tissue. The islands of Langerhans are unaltered (Fig. 371).

When the case is far advanced, the pancreas may become densely sclerotic, fibrous tissue replacing the gland tissue to a great extent, there remaining small masses of the parenchyma embedded in fibrous tissue. The islands of Langerhans are unchanged, being frequently the only remains of the parenchyma, lying amid sclerotic bands. They are resistant to the sclerotic process, and *only suffer in extreme cases when the acini are nearly completely replaced by fibrous tissue.*

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[1] Arch. gen. de. Med., 1900, U. S., iii, 539.

[2] Disease of the Pancreas.

*Chronic Interacinar Pancreatitis.*—With this type of pancreatitis there is a diffuse increase of the interacinar stroma, and the organ is tough, rather than hard, and the surface is smooth. It is characterized by newly formed tissue within the lobules (intralobular) (Fig. 372).

The lesion is irregular in its distribution. There is thickening at one point of the connective-tissue network supporting the acini, while in other regions there are compact bands or masses of stroma. The interlobular tissue is not entirely unaffected, but its proliferation is not constant and is inconspicuous. The lobules are not accentuated, but the interlobular boundaries are obscured by strands of new tissue. The new growth invades the islands of Langerhans.

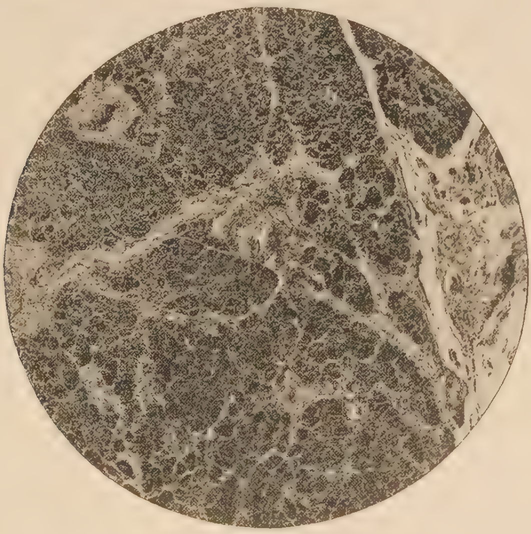

Fig. 371.—Chronic interstitial pancreatitis of the interlobular variety (Santos).

They are almost constantly surrounded by fibrous tissue, forming a capsule which separates them from the adjacent acini, which last are separated from each other. There is proliferation of interstitial tissue about the capillaries of the islands, which forms coarse strands between the columns of cells. In the early stages there is an accumulation of lymphoid cells in and about the islands. In some instances this type of inflammation (sclerosis) is almost entirely limited to the islands of Langerhans.

Interacinar pancreatitis is much less common than the intralobular form, and the former condition has direct association with diabetes mellitus.

Lipomatosis (infiltration with adipose tissue) occurs in association with chronic interstitial inflammation of the pancreas in both types of this lesion.

**Symptoms.**—In many cases the diagnosis of chronic pancreatitis is impossible, particularly in the early stages of the interlobular type of the disease, and at times, even when the lesion is fairly well marked, sufficient of the normal parenchyma remains for the organ to partially perform its functions.

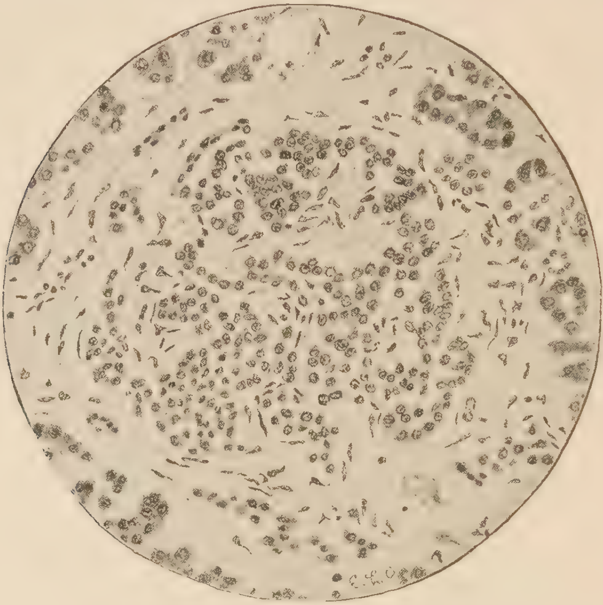

Fig. 372.—Chronic interstitial pancreatitis of interacinar type, showing the invasion of an island of Langerhans by the inflammatory process (Opie).

The *anamnesis* is often of considerable value in arriving at a definite conclusion. A history of bad habits of eating or drinking, with resulting chronic inflammatory conditions of the stomach and duodenum, or of infectious diseases, which are likely to be followed by disease of the biliary passages, or a history pointing to attacks of gall-stones or impacted gall-stone with cholangitis and intermittent fever, would be suggestive of the probability of secondary involvement of the pancreas. In some cases *chronic pancreatitis* may *exactly simulate* this last condition, and the writer will describe a case simulating duodenal ulcer with pyloric stenosis. On the other hand, when with a history as described above there follow, progressive loss of weight, jaundice which tends to persist, though at times it may intermit, disturbances of the digestive functions, pain or aching in the epigastrium, or a sense of discomfort, *an excess of unabsorbed fecal fat*, or *an undue amount of unsplit fat in the feces*, epigastric tenderness, and fulness in this region with or without a swelling of the pancreas, a diagnosis of chronic pancreatitis can be made with certainty. The Cammidge reaction, the writer believes, is not always present, and he, at present writing, is very doubtful as to its value. Jaundice, however, *is not always present.*

**Chronic Pancreatitis Simulating Gastric or Duodenal Ulcer with Pyloric Stenosis.**—Recently the writer examined a patient, a girl of twenty-six years, with a history of gastric disturbances of three years' duration; pain after eating, more frequently after several hours; occasional vomiting; history of apparent occasional attacks of coffee-ground hematemesis. Patient anemic. Total acidity, 60+; free HCl, 30+; combined HCl, 25+. No pus, no occult blood detected in gastric contents. Stool showed some disturbance of intestinal digestion, but nothing typical of chronic pancreatitis. There was local tenderness in one point on the epigastrium. Stomach lies with lower border at umbilicus. The writer believed the case one of pyloric obstruction (partial), due probably to duodenal ulcer. Operation by John Connors disclosed a dilated stomach; no ulcer of stomach or duodenum; *a partial stenosis of the duodenum* from enlarged head of the pancreas; the entire pancreas showed evidences of chronic pancreatitis; no gall-stones in common duct or gall-bladder, but the latter was filled with very thick inspissated bile. The gall-bladder was drained. At present writing, several weeks since operation, there are no gastric disturbances. Whether the head of the pancreas will become smaller, with no subsequent symptoms, or whether the condition later progresses, time alone will determine.

As Deaver justly remarks, the *diagnosis* is most *frequently made* in those who are *driven* to surgical intervention on account of rather an *active type of symptoms.* It seems best to take up the latter separately.

The onset of the symptoms may be apparently sudden, due to an exacerbation, or they may be gradual.

*Pain.*—Pain is usually present during an exacerbation, varying from a dull ache or discomfort to that of a lancinating or colicky type. In the latter instance, calculi are probably responsible in most cases. In many the pain is in the epigastric region, though it may radiate to the back, in the midscapular region. Occasionally it radiates to the right, and it may start beneath the right or left costal margin. There appears to be no definite relation of the time of eating or the character of the food to the commencement of the pain, and this may be sometimes of service in differentiating pancreatic pain from that of gastric or duodenal ulcer.

*Nausea and Vomiting.*—Nausea or vomiting, or both conditions in association, occur quite frequently in attacks. With nausea there may be anorexia, offensive eructations, heart-burn, flatulency, and distaste for fats or meat. Carbohydrates seem to cause in some the most disturbance.

Opie has called attention to persistent vomiting in some cases of chronic interlobular pancreatitis. It seemed to indicate a relationship between gastro-intestinal disturbance and a chronic pancreatitis.

Gastritis or gastroduodenitis may be associated with chronic pancreatitis, and in some cases these factors may be the cause of the vomiting.

Vomiting also occurs with biliary catarrh.

The vomiting of chronic pancreatitis is *not characteristic*, though it may contain mucus and bile, and eructations of distressing character may be present.

*Jaundice.*—Jaundice is another important symptom. In Deaver's[1] cases it was present in 24 patients at the time of operation and absent in 14. In about two-thirds of all cases the common bile-duct passes through the head of the pancreas, so that an inflammation of this part of the organ will interfere with the free exit of bile. Undoubtedly, many cases of so-called catarrhal jaundice can be so explained. On the other hand, a great degree of inflammation of the pancreas may be present, with *no jaundice*, when *the duct does not pass through* the head of the gland. A certain percentage of jaundice cases may be due to obstruction from a calculus or to extension from a catarrhal duodenitis. In some cases the jaundice may come on without pain, and it may be continuous.

In others, rapid wasting and loss of strength are associated with the jaundice, so that *malignant disease of the pancreas may be simulated.* On operation the head of the pancreas may be so hard and nodular as to resemble carcinoma on palpation. Occasionally the neighboring glands may also be enlarged, so that the simulation is complete.

More frequently, jaundice is preceded by pain, sometimes like that of gall-stone colic when such is present, or at other times the pain may be less severe and not colicky.

Pain, intermittent jaundice, and attacks of fever (Charcot's hepatic intermittent fever) may occur, and yet no stone be found at operation. Possibly it has escaped from the duct, though *this simulation of gall-stone attack may occur with chronic pancreatitis.*

The jaundice may vary from a slight tinge to black jaundice.

Opie holds the view that in most cases jaundice is due to cholelithiasis or some other hepatic disease, preceding or accompanying the chronic pancreatitis.

*Digestive Symptoms.*—With serious disturbance of the pancreatic functions there are loss of weight and impairment of appetite. The appetite may persist in some cases. In some there is no disgust for meat. The loss of weight and strength, especially in the cases with jaundice, may be so rapid as to simulate cancer. In ordinary cases marked wasting does not occur.

[1] Jour. Amer. Med. Assoc., April 15, 1911.

*Temperature.*—The temperature is usually normal in prolonged cases, but fever may be present during an exacerbation of the disease. In the cases coming to the surgeon to secure relief, fever has been usually present, sometimes varying from 99° to 103° F. With an associated cholangitis, fever, chills, and sweating occur.

*Bowels.*—The bowels are, as a rule, constipated and flatulence is present. In a few cases there is diarrhea. Only with *advanced pancreatitis* do we have the *frequent pale, offensive, greasy, and bulky movements* (steatorrhea).

**Physical Examination.**—Physical examination may afford no positive information, but may *aid in excluding other abdominal conditions.* Epigastric tenderness and muscular rigidity of the recti in this region may be present during an exacerbation of the disease. When present, it is impossible to detect a mass except possibly under anesthesia. At other times, in patients with thin abdominal walls, or when ptosis of the colon or stomach are present, it may be possible to palpate the enlarged head of the pancreas; but in the *majority* of cases it *cannot be done.* When a mass can be palpated, its relation to the stomach and colon can be determined by inflating the stomach with air or with carbonic acid gas. Resonance on percussion from the overlying stomach, slight movement on deep inspiration, and communicated non-expansile pulsation are characteristic of the mass (enlarged pancreas). The relation of the tumor to the transverse colon can be determined by inflating the colon with air or carbonic acid gas.

*Tenderness.*—Tenderness in the epigastrium is sometimes present, though in some cases it may not be marked. It may be *absent.* Tenderness may be over Mayo Robson's point (above and a little to the right of the umbilicus) or in the midepigastrium. In a number of cases it has been found beneath the right costal margin, but associated disease of the gall-bladder probably accounted for this. Occasionally tenderness is found beneath the left costal margin, and rarely it may be general. If found over the head of the pancreas it is of value.

*Muscular Rigidity.*—This occurs in the epigastrium, though in some cases it may be found in the right hypochondrium, over the gall-bladder. It may be absent.

*Gall-bladder.*—In the chronic cases of long duration a *distended gall-bladder* may be found, such as occurs with carcinoma of the head of the pancreas. With jaundice *due to stone in the common duct* the *gall-bladder* is nearly *always contracted.*

*Liver.*—The liver is found enlarged in some patients. The association of hypertrophic cirrhosis of the liver with interlobular pancreatitis has already been noted.

*The Blood.*—The blood quite frequently shows a secondary anemia which is seldom grave, the hemoglobin also being moderately reduced. Leukocytosis is usually absent except during the acute exacerbation, when it is moderate. The coagulation period should be tested.

When atrophic cirrhosis of the liver and arteriosclerosis accompany diabetes mellitus, the latter condition is due to the interacinar form of chronic pancreatitis, Diabetes with hemachromatosis results from chronic interacinar pancreatitis. One may say that glycosuria and other symptoms of diabetes mellitus are distinctive of chronic interacinar pancreatitis. Temporary glycosuria may occur during an exacerbation of pancreatitis.

*Stools.*—Examination of the stools may give *important information.* In some instances they are clay colored. One must determine to what extent absence of bile influences this condition, and the test for stercobilin must be carried out. Generally the color is due to excess of fat.

In cases of chronic pancreatic disease the total amount of unabsorbed fat may reach 50 to 60 per cent., and up to 90 per cent. with malignant disease of the gland. Müller finds that, though the total per cent. of *fecal fat may not be above normal,* yet the proportion of *split* fat is *always decreased,* averaging about 40 per cent. of the total fat. Katz holds that a diminution of the *split fat below* 70 *per cent.* of the total *fecal fat signifies disease of the pancreas,* except in nursing infants and in patients with severe diarrhea. Steatorrhea is present only in the most advanced cases. Excess of muscle-fibers in the stools is very rare.

*Cammidge Reaction.*—The Cammidge reaction in the urine, in 55 cases of chronic pancreatitis confirmed by Deaver[1] at operation, was present in 18 cases (32 per cent.). In other series the ratio was not as good—one-fifth of the cases positive in pancreatitis as compared with one-sixth positive when no pancreatitis was present; and in summing up all cases in which the condition of the pancreas was determined at operation, Deaver found the pancreatic reaction was obtained about two and a half times as frequently when the pancreas was diseased as when it was not. This does not correspond to the reports of Robson and Cammidge. According to Deaver, in a recent series,[2] he found it present in 25 per cent. The writer is very dubious of the value of the Cammidge reaction. Only recently, in an undoubted case of advanced pancreatitis with *steatorrhea,* repeated tests were negative. The urine should be tested for bile and sugar. Sugar may appear during an exacerbation of an attack and clear up later. *Persistent glycosuria suggests diabetes.* Under Testing the Pancreatic Functions the most desirable methods are described, and those the author prefers are referred to at the end of that chapter, notably the general method of Testing the Intestinal Functions. A marked *hemorrhagic tendency* is of serious import and may show the approach of a fatal termination.

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[1] Jour. Amer. Med. Assoc., April 15, 1911.

[2] Ibid., July 1, 1911.

**Differential Diagnosis.**—With cancer of the head of the pancreas the onset is generally *gradual and painless*, and the age of the patient is usually over forty years. Jaundice[1] appears, which is *absolute*. The gall-bladder is generally *distended*, but is not tender. The liver enlarges from bile stasis, but there are no nodules. Occasionally there is a metastatic tumor of the inner side of the gall-bladder. The loss of weight and strength is rapid and anemia increases. Fever is generally absent and the pulse is slow and feeble. Ascites with edema of the lower limbs may occur late in the disease. Carcinoma is sometimes difficult to differentiate from severe chronic pancreatitis with jaundice. The course of carcinoma is more rapid and the *history short*. Cammidge holds that the pancreatic reaction in the urine occurs in about 25 per cent. of cases of cancer; undigested fat is present in the feces in some cases, of which only a small proportion consists of fatty acids.

When the carcinomatous growth lies in the common bile-duct above the opening of the pancreatic duct, the loss of flesh and strength are not as apt to be as rapid.

*Syphilis.*—This should be excluded by testing for the Wassermann or Noguchi reaction.

*Cancer of the Liver.*—With this condition there is an irregular enlargement of the organ, and it is nodular to the feel. There are a rapid loss of weight and strength, the jaundice is less intense, and there are no fever or paroxysmal pains.

*Gall-stones.*—With gall-stones there is a long history of spasmodic pains without jaundice, the pain commencing in the right hypochondrium and radiating to the right shoulder-blade, as a rule. Later, attacks of pain occur, followed by jaundice, and often recurrent pains, with increased jaundice and with chills and intermittent fever. Head's zone for the gall-bladder is present. In the active stage there is leukocytosis.

The tenderness with gall-stones is over the gall-bladder, and frequently lower down in the *case of pancreatitis* and nearer the middle line. Robson holds that the Cammidge reaction and examination of the stools are of service to exclude pancreatitis. Frequently the *two conditions*—gall-stones and chronic pancreatitis—are associated, and operation alone will determine the presence of the latter. The writer has already referred to the fact that in some cases, chronic pancreatitis presents symptoms of gall-stones alone. Removal of an impacted stone and drainage of the gall-bladder will at times cure the chronic pancreatitis.

With catarrh of the bile-ducts there is at times a history of chronic gastroduodenitis, though it may be of pancreatic origin.

The jaundice and loss of flesh usually come on without pain. With chronic appendicitis, the determination of the sensitive appendix at McBurney's point and of the appendix Head zone are of value.

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[1] The writer reports carcinoma of the tail of the pancreas, with no jaundice and pressure stenosis of the colon with cachexia.

**Prognosis.**—If the diagnosis be made *sufficiently early*, free drainage of the biliary tract, and hence of the *pancreas*, may result in cure.

The disease may progress for months or even for years, and the well-marked cases may *eventually die* from asthenia, diabetes, or the hemorrhagic diathesis. A hemorrhagic *tendency is of bad prognostic import.* Commencing glycosuria, intermitting but increasing in frequency, is suggestive of commencing interacinar involvement; and persistent glycosuria, of diabetes.

**Treatment.**—*Medicinal.*—If gastro-intestinal catarrh is present, proper dietetic measures should be undertaken and the usual treatment accorded to these conditions.

For attacks of gall-stones, olive oil, the salicylates, etc., may be administered. Duodenal or gastric ulcer, alcoholism, syphilis, or arteriosclerosis should receive appropriate treatment if they are believed to be factors. Wet packs, rest in bed, the sparing use of carbohydrates, and the *administration of the pancreatic extracts should be tried.* Mild mercurial purges may be indicated. In all cases, whether or not hemorrhage has occurred for several days before any operation, chlorid or, preferably, lactate of calcium, 60 grains (4.0) a day in divided doses, should be given. It should be continued a week after operation and may be given by rectum, if indicated.

Human serum may be injected hypodermically if hemorrhage occur.

*Surgery.*—For ulcer of the stomach or duodenum—if these are factors—gastro-enterostomy may be required. Free drainage of the biliary tract and, through this, drainage of the pancreatic ducts will in many cases enable the pancreas to cast off the infection and resume its normal functions. Gall-stones should be removed from the gall-bladder and the common bile-duct as well, and the head of the pancreas should be thoroughly explored, and any calculus lying therein or in the pancreatic duct should be removed.

*Cholecystotomy* or, in some cases, cholecystenterostomy (establishing an artificial opening between the gall-bladder and duodenum) afford the best method of drainage. Junction of the gall-bladder with the colon, the writer believes, increases the liability of infection.

The writer's case, with colon bacilluria, received urotropin and sodium benzoate and the usual treatment of colon bacillus infection as described in that section.

*Diet.*—If temporary glycosuria or diabetes mellitus be present, appropriate diet should be instituted. With the interlobular type of pancreatitis fats should be given in the form of emulsions and milk is of great value. Feeding should be forced by repeated small meals and general nutrition should be improved. The bowels should be regulated to one movement daily and diarrhea checked.

## CHAPTER XLI

# FAT NECROSIS—TUBERCULOSIS—SYPHILIS—PANCREATIC CALCULI

### FAT NECROSIS

BALSER[1] first directed attention to this condition. In several autopsies he observed in the fat about the pancreas, small white opaque areas, which subsequently were demonstrated to be composed of necrotic fat cells. One occasionally finds these minute foci limited to fat within or upon the pancreas, but usually the process is disseminated.

**Location and Appearance of Fat Necrosis.**—Fat necrosis is of importance to the surgeon, as it indicates disease of the pancreas. When the abdomen is opened, the omentum, and at times other fat, is found to be studded with small round oval areas, opaque, white or yellow in color, in contrast to the translucent golden yellow color of normal fat.

Occasionally each area of necrotic fat is surrounded by a narrow hemorrhagic zone.

The lesions are usually most marked near the pancreas, and are generally limited to the fat of the abdominal cavity. Two cases of fat necrosis in the subcutaneous tissue of the abdomen have been reported by Hausemann.[2] Areas of fat necrosis have been found in the mesentery, in the subperitoneal fat of the abdominal wall, in the subpericardial and subpleural fat, and in the pericardial fat.

**Occurrence.**—Fat necrosis associated with pancreatic disease has been frequently noted in stout patients, in fact in about 50 per cent. of cases. In the majority it is found with hemorrhagic necrosis or gangrene of the pancreas, is rare with suppurative pancreatitis, and occasionally occurs with chronic pancreatitis. It may be present with pancreatic calculi, cyst of the pancreas, and with gall-stones or carcinoma which close the pancreatic duct. Fat necrosis with acute hemorrhagic pancreatitis is depicted under that subject.

**Pathology.**—The changes in the fat cells are due to splitting of the neutral fat into fatty acid and glycerin. The fatty acids are deposited in needle-like crystals in the cells. The latter lose their nuclei and are necrotic. The soluble glycerin is absorbed. The fatty acids subsequently unite with calcium and form irregular or globular masses of lime salts. The outline of the cells is more or less preserved.

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[1] Ueber Fettnekrose, Virchow's Archiv., 1882, xc, 520.

[2] Berliner Med. Gesellsch. Sitzung vom 4 Dec., 1889; Berliner Klin. Woch., 1889, xxv, 1115.

It has been demonstrated by Benda[1] that acetate of copper will combine with the fatty acid in the necrotic fat, and will produce a greenish-blue compound, and that calcium salts are less markedly stained, while neutral fats are unchanged. It is thus possible to demonstrate areas of necrosis which are not visible to the eye. A half-saturated solution of acetate of copper is employed for this test.

The foci of fat necrosis may disappear entirely. This has been demonstrated by subsequent operation on patients in whom they were formerly observed.

**Etiology of Fat Necrosis.**—Some have attempted to impute the occurrence of fat necrosis to the action of micro-organisms, since the colon bacillus and various micrococci have been found. The invasion of the fat by these bacteria is believed to be secondary.

*The products of the pancreas are the active cause of fat necrosis.*

Flexner,[2] Opie,[3] and others have demonstrated on animals that when conditions were produced which afforded opportunity for the escape of pancreatic secretion into the tissues surrounding the gland, fat necrosis resulted.

It has been concluded that fat necrosis is produced by the fat-splitting ferment of the pancreatic juice.

Flexner[4] has demonstrated that necrotic fat tissue in the human being contains an enzyme which can split up neutral butter fat and set free fatty acids.

Some have claimed that fat necrosis may occur without lesion of the pancreas. A case associated with duodenal ulcer is reported in this volume. The presence of an aberrant pancreas may explain some cases. Fat necrosis without the association of pancreatic disease is a rare occurrence.

Occasionally (postmortem) minute foci of fat necrosis are found in and upon the pancreas.

The treatment of fat necrosis when determined at operation is that of the pancreatic disease upon which it depends.

## TUBERCULOSIS OF THE PANCREAS

Tuberculosis of the pancreas occurs, as a rule, in connection with tuberculosis of other organs, and the blood-vessels usually furnish the channel by which the bacilli are distributed to the gland. In 12 out of 128 autopsies with tuberculosis in other regions, Krudewetzky[5] found miliary tubercles of the pancreas, of which 6 were in children in 18 cases of acute miliary tuberculosis. The tubercles usually lie within the lobules, and less frequently in the interlobular tissue.

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[1] Virchow's Archiv., 1900, clxi, 194.

[2] Contributions to the Science of Medicine, Johns Hopkins Hosp. Reports, 1900, ix, 859.

[3] The Pancreas.

[4] Jour. of Exper. Med., 1897, ii, 413.

[5] Zeit. f. Heilk., 1892, xiii, 101.

They rapidly undergo caseation. In cases of chronic tuberculosis large tubercles occasionally occur.

Multiple small tuberculous deposits are found irregularly scattered through the pancreas, or several large masses, which may caseate and form cavities that open into the stomach or some adjacent organ. A small tuberculous lymphatic gland has been removed from the head of the pancreas. Tuberculosis of the pancreas may occur by extension from neighboring organs.

Primary tuberculosis of the pancreas, though rare, has been reported by Mayo,[1] Aran,[2] and Chvostek.[3]

*Symptoms.*—Pain in the epigastrium, vomiting, local tenderness, and, at times, jaundice may be present. A palpable tumor just above the umbilicus was found by Mayo in 1 case. Functional disturbances of the organ may also occur. The diagnosis is probable from the usual association of the process elsewhere.

## SYPHILIS

**Congenital Syphilitic Pancreatitis.**—Attention was first called to the frequency of congenital syphilis of the pancreas by the investigations of Birch-Hirschfeld,[4] who found it in that organ in 13 out of 23 children, and in another series in 29 out of 124 syphilitics. The pancreas would seem to be affected in about 22 to 23 per cent. of all cases of syphilis of the newborn.

*Pathology.*—The pancreas is enlarged and firm, and there is a diffuse interstitial pancreatitis with proliferation of interlobular and interacinar tissue which penetrates between the cells of the acini. This is followed by destruction of the parenchyma, which last probably atrophies. There is a syphilitic peri-arteritis. The islands of Langerhans are not invaded by the new growth of interstitial tissue. Small gummata (syphilitic necrosis) are occasionally found in the newly formed stroma.

**Acquired Syphilis.**—Gummata and syphilitic induration, resembling syphilitic cirrhosis of the liver, may occur. The bands of tissue are irregular, scar-like, and differ from the diffuse increase of tissue, such as occurs with the interlobular or interacinar types of chronic pancreatitis.

*Symptoms.*—Usually one finds associated symptoms of the tertiary stage in these acquired cases. These may be disturbances of the pancreatic functions, evidences of chronic pancreatitis, and, at times, diabetes.

*Diagnosis.*—The Wassermann or Noguchi reactions should be tested for in addition to a careful anamnesis and examination.

*Treatment.*—This should be as of syphilis—mercury and iodids—or, preferably, "606" should be injected.

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[1] Cited by Senn, Outlines of Human Pathology.

[2] Ibid.

[3] Wiener med. Blätter, 1879, ii, 791.

[4] Gerhardt's Handbuch d. Kinderkrankheiten, iv, Abt., 753, Tübingen, 1880.

## PANCREATIC CALCULI

Pancreatic calculi are rare, and among 1500 autopsies at the Johns Hopkins Hospital only 2 cases were discovered. The earliest report of this condition is by Panarol and Galea in the year 1667.

Guidiceandra, in 1896, records 48 cases; and in 1903 Oser states that there were only 70 cases reported. Lazarus has since collected 80 cases. Mayo and Robson have reported others since that date.

**Occurrence.**—Pancreatic calculi are more common in men than in women. Lazarus[1] collected 57 cases, of which 47 occurred in men and 10 in women.

**Age.**—Thirty-seven of these cases were in patients between thirty and forty years of age.

**Pathology.**—Calculi are not found in the healthy pancreas, and probably these pancreatic concretions are a result of catarrh of the ducts, with stagnation of secretion, with which bacterial infection is associated.

The concretions in the ducts may be like sand, but generally there are one or more small stones. As many as one hundred have been reported. The ducts may be lined with calcareous material, so as to completely or nearly close their lumen.

Usually the calculi consist of calcium carbonate or calcium phosphate, though a case has been reported which contained cholesterin in considerable amount, and another which consisted chiefly of calcium oxalate.

Biliary calculi at times make their way into the pancreatic duct, and this has resulted in suppuration. A pancreatic calculus which lodges at the duodenal end of the pancreatic duct may be stained with bile-pigments and be covered with a layer of cholesterin. The duct of Wirsung is narrowest where it passes through the *wall of the duodenum*, and calculi have a tendency to lodge *above this point.*

The outflow of the pancreatic secretion may be entirely obstructed, and the duct may become dilated above the point of obstruction. True cysts rarely result, but they have been reported. Obstruction of the duct by calculi is usually associated with bacterial infection, and produces the most advanced type of sclerosis. The Bacillus coli has been found in two calculi. The chronic inflammation is of the interlobular type, and the islands of Langerhans are usually not affected. They may finally become involved in the advanced sclerosis.

Pancreatic calculi are opaque to the *x*-rays, and we *thus have a means of diagnosing their presence* and of differentiating them from biliary calculi, which are not readily seen and may not even be visible in a röntgenograph. The presence of the Cammidge reaction, when such is present, Robson believes, will confirm the diagnosis by demonstrating the associated chronic pancreatitis.

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[1] Beiträg zur Pathologie und Therapie der Pankreaserkrankungen mit bensonderer Berucksichtigung der Cysten und Steine, Berlin, 1904.

The stones are round, oval or elongated, and usually smooth. They may occur in all parts of the ducts, though they are found most frequently in the head of the pancreas. They may also be branched.

One stone has been recorded measuring 2½ by ½ inch, weighing 200 grains, and a second one weighing 20 ounces. They are pale in color, but may be white, or when near the common duct may be covered with cholesterin and stained with bile.

Obstruction to the flow of the pancreatic juice from an impacted calculus may cause escape of the secretion into the tissues, so that fat necrosis results. A pancreatic calculus in the diverticulum of Vater may occlude the common bile-duct and cause jaundice. Ulceration of the mucosa of the duct may be caused by an impacted calculus, and pancreatic abscess result.

**Etiology.**—The writer has already referred to the fact that obstruction to the outflow of the pancreatic juice with associated bacterial infection are probably factors. Various forms of bacteria have been found within the calculi. Cholelithiasis may occur in association with pancreatic lithiasis, and the former may produce obstruction of the pancreatic duct and favor stasis. Pancreatic calculi are sometimes found in pancreatic cysts, possibly resulting from the retained secretion.

**Symptoms.**—Pancreatic calculi have been found on autopsy in persons who have never suffered with symptoms that could be referred to the pancreas. In some the symptoms may be vague. The presence of a cyst, a secondary abscess, and the degree of advancement of the chronic pancreatitis modify the symptoms.

There is often pain in the epigastrium, which may be slight but continuous, or it may be paroxysmal, resembling biliary colic; sometimes it is agonizing in character, and may be associated with vomiting, hiccup, cold sweats, and collapse; chills and fever may at times occur. The attacks may be brought on by exertion, or they may be irregular, occurring at any time, day or night. The pain frequently comes on in sharp, colicky attacks, less severe than gallstones, and may radiate toward the inferior angle of the left scapula. A sensation of soreness or stiffness is sometimes noticed for several days after an attack. Fragments of stone may subsequently be found in the stool, which, when analyzed, consist chiefly of carbonate or phosphate of calcium. In 1 case of Kinnicutt's[1] the pain began in the back and then ran around the right side along the lower intercostal spaces, while in another case the pain began between the scapulæ and ran through—not around—the body into the epigastrium, where it became localized to the right of the middle line. The localization of the pain, though more frequently to the left, is not always characteristic.

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[1] Trans. Assoc. Amer. Phys., 1902, xvii, 81.

Jaundice, which occurs with pancreatic calculi, may be due to associated gall-stones, but a pancreatic calculus may occlude the common bile-duct and thus produce it. Lancereaux records glycosuria in 12 out of 40 cases of pancreatic calculi. Sugar may be observed at intervals. Diabetes mellitus or glycosuria accompany pancreatic lithiasis with a fair degree of frequency, and Lazarus has found one of these conditions present in 36 out of 80 cases.

The disturbance of carbohydrate metabolism is not produced by the occlusion of the ducts, but by the chronic interlobular pancreatitis, and it occurs only when this condition is so far advanced that the islands of Langerhans are involved.

Alimentary glycosuria indicates a less advanced stage of the pancreatitis.

Steatorrhea, when it is present, is an aid to diagnosis. It has been recorded in only 10 out of 80 cases. Even if there is no apparent excess of fat, microscopically, in the stool, there may be *deficient fat splitting of ingested fats* into *fatty acids and soaps*, and an excess of neutral fat may be found.

Azotorrhea is less frequently found.

Suppuration, as already noted, may occur as a complication of pancreatic calculus.

The *hemorrhagic tendency* may occur with pancreatic calculi, especially with advanced chronic pancreatitis.

**Diagnosis.**—Pancreatic calculi are more *opaque for x-rays than are gall-stones*, and this fact proves of value for diagnosis.

The *analysis of the calculus, calcium carbonate or phosphate, when present in the stools*, is also of use. Glycosuria, when present, even though temporarily so, is significant, and also deficient splitting of the ingested fats into fatty acids and soaps. Robson and Cammidge set much store by the pancreatic reaction of the urine, which they believe usually to be present, but the writer believes it uncertain. The location of the pain and the direction of its radiation in some cases proves of service.

**Treatment.**—Lazarus recommends that secretion be increased by the administration of large quantities of water, and especially to acidify it with carbonic acid gas or weak acids. Pancreatic secretion may be increased by the injection hypodermically of pilocarpin, $\frac{1}{100}$ grain (0.0006), and Eichhorst reports a favorable result. This method may be dangerous, particularly if there is obstruction in the duct.

Possibly prosecretin, 3 grains (0.5), which is less active and a normal excitant, three times a day, might be of service. Morphin, $\frac{1}{8}$ to $\frac{1}{3}$ grain (0.0075–0.0022), may be administered by hypodermic for the acute attacks of pain; heat should be applied to the epigastrium, and shock be treated in the usual method, if such be present. A hot bath is useful, as in attacks of renal or biliary colic.

*Surgery.*—In several cases pancreatic calculi have been successfully removed by operation through incision in the duodenum or, preferably, by pancreolithotomy.

It is indicated to explore the gall-bladder and common bile-duct and remove calculi, if present. If there is hemorrhage from the gastro-intestinal tract or subcutaneous or nasal hemorrhage, and, in any event, for several days before operation and for at least a week thereafter, calcium lactate should be administered in divided doses, 60 to 90 grains (4.0–6.0) per day by mouth or by rectum, if there is vomiting. Hypodermic injection of normal human serum, or horse serum by mouth, may be further indicated for hemorrhage.

## CHAPTER XLII

# CYSTS OF THE PANCREAS—NEOPLASMS

### CYSTS

Cysts of the pancreas, though not frequent, must be considered in the diagnosis of cysts of the abdomen, since they occur in various regions, and may simulate other diseases.

About 173 cases of operation on cysts of the pancreas have been recorded. Hale White states that in nearly 6000 autopsies at Guy's Hospital from 1883 to 1894 only 4 cases of pancreatic cyst were found, 1 of which was a hydatid cyst.

The simplest classification is as follows:

1. True cysts of the pancreas.
2. Pseudocysts.
3. Hydatid cysts.

1. Among the *true cysts* we have:

(*a*) Retention cysts, due to obstruction of the outflow of the pancreatic secretion, with resulting dilatation of the ducts of the acini.

(*b*) Proliferation cysts, or cystic neoplasms. These are cystic tumors, due to spontaneous proliferation of the epithelial elements of the gland, with accumulation of fluid in the cavities which are so formed. They may be simple, such as cystadenomata; or malignant, cystic epitheliomata or carcinomata.

(*c*) Congenital cystic disease of the pancreas is exceedingly rare.

**Retention Cysts.**—*Etiology.*—Partial or intermittent occlusion of the pancreatic duct favors the formation of retention cysts of the pancreas.

The outflow of pancreatic secretion may be interfered with by obstruction of the excretory duct, or by compression from without and obstruction within it.

Compression of the pancreatic ducts by tumors, by gall-stones, or closure by a pancreatic calculus, or by a large calculus in the diverticulum of Vater, tumors in the bile-passages or duodenum, swollen lymphatic glands pressing on the duct, adhesions or *intestinal parasites* in the pancreatic duct, may be causes. Frequently no cystic formation results, but sometimes cysts are found.

Chronic pancreatitis is believed to be the most common cause of retention cysts.

Occlusion of the ducts is followed by interlobular pancreatitis, which, when far advanced, may cause diabetes mellitus.

Retention cysts are frequently quite small, and may not cause symptoms. There may be a beaded or rosary-like dilatation of the pancreatic duct, which has been described as "ranula pancreatica," or multiple cysts of small size containing opaque thick fluid, "acne pancreatica," or a single cystic dilatation as large as a man's fist.

They are occasionally multiple—two of equal size, unilocular or multilocular. One cyst may have several small cysts attached to its walls.

When a cyst enlarges it destroys the substance of the gland, and, at the same time, interstitial inflammation is produced in the adjacent gland-tissue. The cyst may become pedunculated. Retention cysts of large size have been reported containing 15 to 20 quarts (liters) of fluid.

The wall of the cyst is composed of dense fibrous tissue with few cells, and may be 3 to 4 mm. thick. Occasionally the wall is thin. Large blood-vessels often traverse the outer surface.

The inner surface consists of an epithelial lining of a single layer of cylindric cells, which may be flattened by pressure. It is sometimes smooth and shining, and free from epithelium.

In some cases the cyst lining may have ridges or septa; the remains of other cysts or clotted blood may be found therein adherent to the surface. Portions of pancreatic tissue are sometimes embedded in their walls.

**Proliferation Cysts, or Cystic Neoplasms of the Pancreas.**—Many of these tumors are on the border-line between proliferating cystoma and cystic carcinoma, and only the subsequent course of the tumor will indicate to which class it belongs. They are characterized by irregular gland-like growths below the epithelial lining of the cyst and by the presence of papillary projections from the cyst lining. There are two types—the cystadenoma and the epitheliomata cysticum.

The simple proliferation cyst (cystadenoma) is usually multilocular, has a lining of columnar epithelium, which sometimes dips down into the wall of the cyst, and at times covers polypoid masses, which project into its cavity.

These cysts are more common in the tail of the pancreas, and often their contents are blood stained.

Rarely cystic tumors of the pancreas are malignant (the cystic epithelioma). Hartmann[1] described one of the tails of the pancreas which was accompanied by metastases in the liver.

Sotti, under the name of adenocystoma papilliferum, described a tumor of the pancreas with metastases in the lymphatic glands and lungs.

The malignant form is usually multilocular, with patches of carcinomatous tissue in its walls.

In some cases chocolate-colored fluid is contained in the cyst.

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[1] Cong. franc de Chir., 1891, v, 618.

Metastatic deposits occur in the liver, pancreatic glands, duodenum, and in other organs, rendering the tumor inoperable.

Congenital cystic disease of the pancreas is exceedingly rare. Robson and Moynihan refer to 3 cases, resembling that met with in other organs.

Hydatid cysts of the pancreas seldom occur resembling the lesion met with in the liver. Evacuation and drainage are indicated.

**Pseudocysts of the Pancreas.**—Pseudocysts constitute a large proportion of cases reported as pancreatic cysts. They may be found within the substance of the pancreas, but the greatest number of pseudocysts are formed in contact with the gland, particularly *in the lesser peritoneal cavity*, as a result of injury to the pancreas.

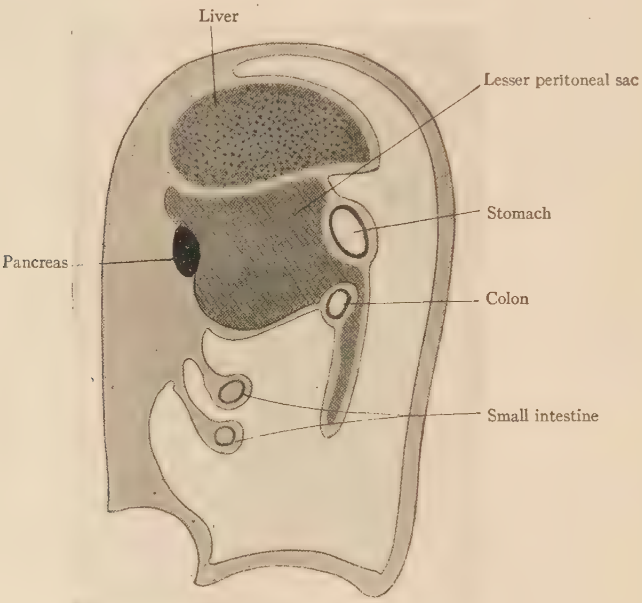

Fig. 373.—Diagram to show the method of origin of a pseudocyst of the pancreas (Robson and Cammidge).

Blood and pancreatic juice escaping into the lesser cavity set up a mild form of peritonitis, which closes the foramen of Winslow and produces a tumor which is difficult to differentiate from a true cyst. Enzymes similar to those of the pancreatic juice have been found in traumatic cysts. Fig. 373 is shown the method of the origin.

Pseudocysts within the pancreas containing blood have been believed to be the result of hemorrhage into the organ.

The wall of these cysts is formed by dense connective tissue. There is no epithelial lining to the cyst, but this does not always determine the nature of the lesion, since the epithelial lining may be destroyed by the pancreatic juice.

*Etiology.*—In 33 out of 117 cases of pancreatic cyst, Körte has found that traumatism to the abdomen preceded the appearance of a palpable tumor. Lazarus has collected 8 more cases.

Among the various injuries to the epigastric region are the kick of a horse or compression between car buffers. Indirect force may produce it also, such as a violent fall, by tearing the pancreas.

These traumatic pseudocysts usually contain blood, though only clear fluid be present.

*Hemorrhagic necrosis of the pancreas* may be the direct cause of pseudocysts. A number of such cases have been reported, the patient having recovered from the attack of acute pancreatitis, with the subsequent appearance of the cyst.

*Sex.*—The greater portion of the traumatic pseudocysts occur in males, who are most exposed to injury, while true cysts occur more frequently in females.

**Cystic Contents.**—The fluid in the pancreatic cysts and pseudocysts may furnish little evidence as to their origin. Blood is generally present. The fluid is frequently reddish brown; it may be coffee-colored, yellow, greenish, milky, black, or bright red from recent hemorrhage. After absorption of the blood, traumatic cysts may contain clear fluid. The contents are sometimes viscid, more or less turbid, and mucin has been demonstrated therein.

The specific gravity varies from 1.007 to 1.028, and the reaction is generally alkaline, rarely neutral, and still more rarely acid. Sugar is seldom met with. Albumin is present. Under the microscope, red blood-cells, fat-cells, epithelial cells, crystals of fatty acids, leukocytes, necrotic tissue at times, at times cholesterin, and, rarely, leucin and tyrosin are present.

One or more of the pancreatic enzymes can frequently be demonstrated in these cysts. Proteolytic, lipolytic, and diastatic enzymes have been found in the fluid of abdominal cysts which did not originate from the pancreas, and cysts of the pancreas may show no activity of these enzymes. Nevertheless, if a cyst contains fluid which can digest coagulated egg-albumen or split starch, it is probable that it originates from the pancreas.

**Symptoms.**—In studying the symptoms of the pancreatic cyst one must consider the possible cause from which the cyst may develop, the general symptoms, both subjective and objective, the physical signs of the tumor, and, finally, the pressure symptoms which may result from its presence.

There may be a history of previous traumatism in the epigastrium or of an attack of hemorrhagic necrosis of the pancreas, subsequently followed by the development of a tumor, or of previous attacks pointing to pancreatic calculi, or disturbances suggestive of a chronic pancreatitis.

The *Röntgen rays* may be of service in determining the presence or *absence of pancreatic calculi*, also the examination of the stool. The last may also demonstrate disturbance of the pancreatic functions.

On the other hand, cases do occur which have existed for a long time and present *few or no symptoms except the presence of the tumor.*

As regards general symptoms, pain in the epigastrium or just above the umbilicus may be present, and may occur even when the cyst is not palpable. There may be colicky paroxysms, varying in location and intensity, sometimes there is *no pain* whatever. Vomiting may occur, associated with the acute attacks or pain, or the cyst may be bound to the stomach by adhesions and produce gastric disturbance. Constipation is usually present. Bulky pale motions are rare and only occur with advanced pancreatitis. The cyst may compress the lumen of the intestines and produce intestinal obstruction. Jaundice does not occur as frequently with pancreatitis cyst as it does with carcinoma, because the former is less frequently in the head of the gland.

In some cases disturbances of digestion referable to a diminution of the flow of pancreatic juice into the intestines occur. Fitz has found only 2 cases of steatorrhea with cyst of the pancreas, and only 2 in which there were undigested muscle-fibers in the feces. These last conditions show advanced destruction of the gland. Glycosuria is associated with them.

Glycosuria and diabetes mellitus follow severe chronic inflammation of the pancreas, which last is produced by a cyst occluding the ducts. Diabetes was present in only 9 out of 134 cases which were collected by Oser. Alimentary glycosuria has been reported.

*Emaciation* is a frequent symptom, and in some cases cannot apparently be explained as due to disturbances of pancreatic digestion. The patient frequently rapidly regains weight after drainage of the cystic contents.

Robson and Cammidge report marked pancreatic reaction in the urine. The writer believes this of no value.

*Pressure Symptoms.*—Among the symptoms due to pressure of the cyst are: Dilatation of the superficial branches of the portal vein, ascites from pressure on the portal vein, or edema of the lower extremities from pressure on the inferior vena cava.

In 2 cases the right ureter has been obstructed. Dyspnea may result from pressure on the diaphragm or from distention of the abdominal cavity. Rarely, intestinal obstruction may result from pressure.

**Physical Signs of Pancreatic Cysts.**—The cyst is usually spheric and the surface is smooth. It may be as large as a human head, and may even fill the entire abdominal cavity from the ensiform cartilage to the symphysis. Usually fluctuation can be felt, but at times the sac is so tense that it appears to be a solid growth.

The tumor is *often situated in the middle line between the ensiform cartilage and the umbilicus.* It may cause a round protrusion of the abdominal wall; in *many instances the greater part of the cyst lies to the left of the median line*, and in a few cases it was found on the right side. Occasionally cysts have extended to below the umbilicus.

Usually these cysts exhibit little mobility unless they are in contact with the diaphragm, in which case they move with respiration. When they are situated in the tail of the pancreas they may be freely movable. If the cyst is in contact with the aorta, pulsation may be transmitted, but it disappears when the patient is in the *knee-chest* position.

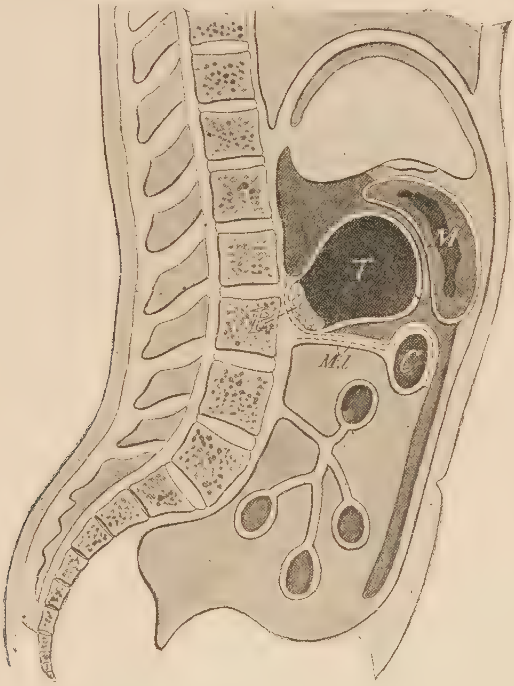

Fig. 374.—A cyst projecting from the ventral surface of the pancreas into the bursa omentalis. The stomach is in front of the cyst and with its growth is pushed upward; the transverse colon is below it: *M*, stomach; *C*, transverse colon; *P*, pancreas; *M.t.*, transverse mesocolon.

The relation of the different parts of the pancreas from which the cyst originates to adjacent organs determines the physical signs.

The relation of the cyst to the stomach and colon can be defined after artificial distention of these organs with carbonic acid gas or with air. Körte studied 133 cases of pancreatic cyst which were operated upon, and classified the positions which they occupied.

Pancreatic cysts usually project from the anterior surface of the pancreas into the lesser peritoneal cavity and push the stomach upward. They are covered by the gastrocolic omentum (Fig. 374).

Small cysts lie behind the stomach.

Cases have been reported in which the cyst arises from the upper border of the pancreas, and makes its way between the stomach and liver, being covered by the gastrohepatic omentum (Fig. 375).

The dulness of the tumor is continuous with the dulness of liver. Inflation of the stomach will cover the dull area of the tumor.

In some the cyst grows between the two layers of mesocolon, and the cyst projects upon the upper surface of the mesocolon and lies between the stomach and colon (Fig. 376).

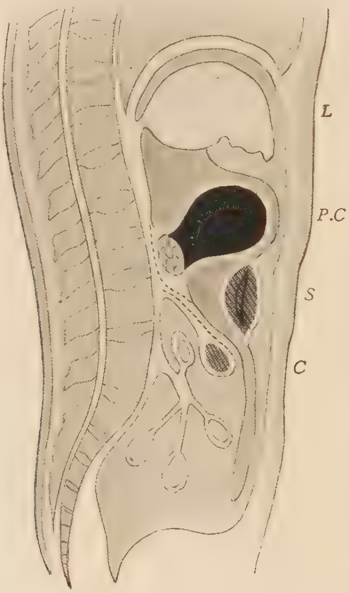

Fig. 375.—Pancreatic cyst between liver and stomach (Robson and Cammidge): *L*, Liver; *P.C*, pancreatic cyst; *S*, stomach; *C*, colon.

Occasionally, the cyst may project on the under surface of the mesocolon and the transverse colon lie above it (Fig. 377), or the colon may cross the summit of the cyst (Fig. 378).

The cyst may pass into the right hypochondrium and simulate an enlarged gall-bladder, or right renal, or suprarenal, cyst. It may make its way into the right or left lumbar region and simulate a cyst of the kidney; or it may pass forward to the right and beneath the hepatic flexure of the colon and resemble a tumor of the kidney, cecum, or ascending colon. In some instances it burrows between the layers of the mesentery, simulating a mesenteric cyst, or may pass to the left and resemble a left renal or ovarian cyst, or a cyst of the

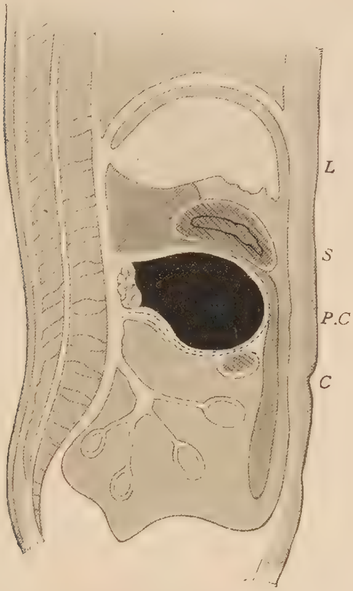

Fig. 376.—Pancreatic cyst between stomach and colon (Robson and Cammidge): *L*, Liver; *S*, stomach; *P. C*, pancreatic cyst; *C*, colon.

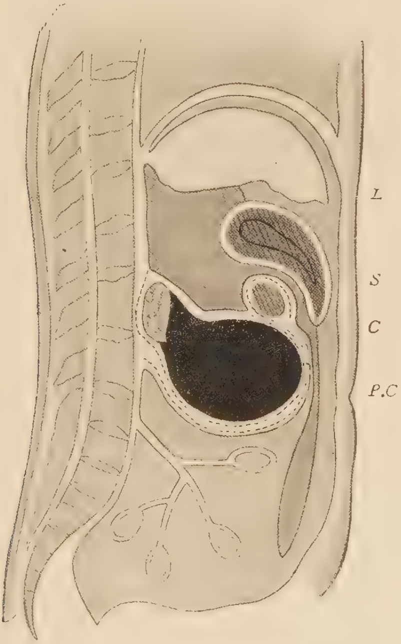

Fig. 377.—Pancreatic cyst below the transverse colon: *L*, Liver; *S*, Stomach; *C*, colon; *P.C*, pancreatic cyst (Robson and Cammidge).

spleen or left lobe of the liver. It has also simulated a tumor of the descending colon or of the small intestine.

Accumulation of fluid in the lesser peritoneal cavity (pseudocyst) may closely resemble the true pancreatic cyst. A cyst may pass through the foramen of Winslow into the general peritoneal cavity, or may rupture into the peritoneal cavity as the result of injury produced by a blow or fall.

The spontaneous disappearance of a cyst without intraperitoneal rupture has been reported in association with diarrhea. The contents probably entered the intestine. Bull[1] and Parsons report such cases. Diarrhea occurred at the time of the disappearance. Possibly one of the pancreatic ducts communicated with the cyst.

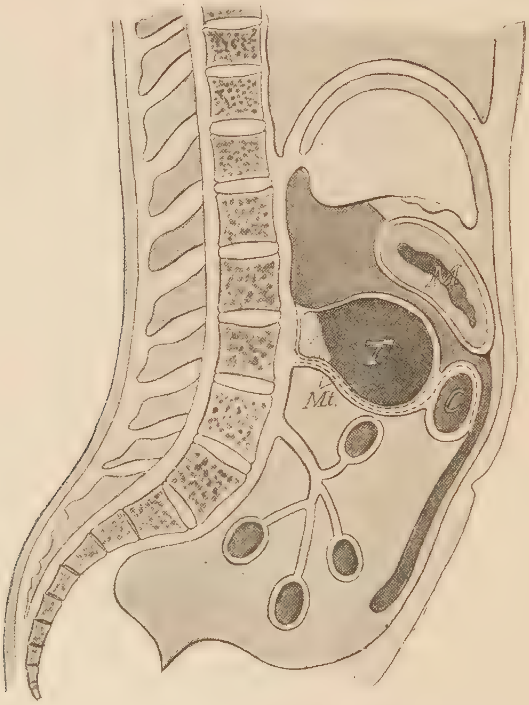

Fig. 378.—A pancreatic cyst (*T*) which has penetrated between the folds of the transverse mesocolon (*M.t.*) and projects equally upon the upper and lower surface. The stomach is above the cyst and the transverse colon crosses it.

**Diagnosis.**—A round fluctuating tumor *lying in the epigastrium, especially when the greater part is to the left of the median line*, is suggestive of a pancreatic cyst. Most frequently the cyst lies between the stomach and transverse colon. The stomach usually partially covers it. Even when the cyst occupies the more uncommon position between the stomach and liver, percussion of the cyst while inflating the stomach is of value, since tumor gradually becomes covered as the stomach distends. Cysts of the *liver and spleen* or a *distended gall-bladder* remain *superficial*, and *are not pushed back* and covered by the distending stomach.

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[1] N. Y. Med. Jour., 1887, xlvi, 376.

Mesenteric cysts usually lie near the umbilicus and are *freely* movable. Pancreatic cysts may appear in this region, but generally there is *no mobility*. At times the pancreatic cyst may extend as far as the pelvis.

The history of the cyst as first commencing in the epigastrium may be of service; also inflation of the colon may aid in the determination of its relations, and bimanual examination of the vagina and rectum may exclude its connection with the uterus and ovaries.

The disappearance of the tumor is more suggestive of hydronephrosis.

Exploratory aspiration of the cystic tumor the writer believes dangerous. It may damage the viscera or cause extravasation of the cystic contents.

The history is of great value in many cases.

The Cammidge reaction may be tested, though the writer has no faith in the procedure. The pancreatic functions should be investigated and the urine examined for sugar. These factors are an aid to diagnosis. The contents of the cyst should be tested for enzymes after evacuation by operation.

**Treatment.**—Surgical treatment is alone of service. Aspiration is dangerous for the purpose of diagnosis. Incision and drainage is the operation of choice; extirpation is more dangerous, and in many cases is impossible. Description of the surgical procedures has no place in this volume.

It is recommended to administer a diet to lessen the flow of pancreatic juice, thus, bread and milk, with the administration of small doses of sodium carbonate, 5 to 10 grains (0.32–0.65) t. i. d., are excellent. Others advise a diabetic diet and the use of the same drug.

As in all cases of pancreatic disease, chlorid or, preferably, lactate of calcium, 1 dram (4.0) in divided doses, should be administered for a week before and after operation. In the latter event it can be given for a time by rectum. Human serum may be used by hypodermic, and the calcium salts, 60 to 90 grains (4.0–6.0) daily, by mouth, if hemorrhage occur at any time during the course of the disease.

Mayo Robson records 160 cases of pancreatic cyst which have been operated. Of these, there were 20 deaths subsequent to operation, and 8 deaths later from diabetes or other complications. Of 138 cases treated by incision and drainage, death occurred in 16. Among 15 cases in whom complete excision was practised, 3 died. Partial excision in 7 cases resulted in 1 death. Körte collected 102 cases, of which 101 were treated by incision and drainage, with only 4 deaths.

## NEOPLASMS

Tumors of the pancreas are not a very common occurrence, and are most frequently of a malignant nature. The solid growths met with in the pancreas are carcinoma, sarcoma, adenoma, and lymphoma. Segré,[1] in 11,472 autopsies, reports 127 cases of carcinoma and 2 of sarcoma. Benign tumors are less common.

### CARCINOMA

Carcinoma is the most common of the new growths found in the pancreas. Probably some cases reported as cancer of the liver were cases of primary carcinoma of the pancreas, with secondary nodules in the liver. Formerly cancer was considered the most common lesion affecting the pancreas, but chronic interstitial pancreatitis was undoubtedly mistaken for it in many instances.

**Age.**—Carcinoma usually occurs after forty years of age, though a case in a patient aged thirty-two has been reported, and rarely it has occurred in childhood.

**Sex.**—Carcinoma is more frequent in males. In 108 cases[2] there were 69 in men and 39 in women.

**Situation of Tumor.**—Carcinoma is most frequently situated in the head of the gland. It was involved in about 62 per cent. of the cases recorded: in 5.5 per cent. the tail was affected; in 3.5 per cent., the body; and there was a diffuse growth in 29 per cent. The duct of Wirsung was obliterated in 55 out of 66 cases, and dilated beyond the point of stricture in about one-third of the cases. Among 57 cases collected by Segré, the tumor was situated in the head of the gland in 35 cases; in the body, twice; in the tail, once, and the entire gland was affected in 19 cases. Out of 113 cases reported by Miraillié, primary carcinoma of the head of the pancreas was present in 82.

Oser states that there is secondary carcinomatous involvement of the pancreas in more than 10 per cent. of cases of primary carcinoma of the stomach. Metastasis also takes place from carcinoma of other organs.

**Frequency.**—Out of 53,000 autopsies, Roswell Park reports 226 cases of primary malignant disease of the pancreas. Secondary growths of the pancreas are much more common. Eppinger reports, in 1314 autopsies, 19 cases of cancerous involvement of the pancreas. Only 2 of these were primary cancer of the pancreas. There were 308 cancers in various organs.

**Morbid Anatomy.**—Carcinoma of the pancreas begins either in the glandular epithelium or in the cells of the excretory ducts. In the former instance it is of the spheroid type, and in the latter usually columnar.

Spheroid-celled carcinoma is the more common and usually of the

[1] Annual Univ. de Med. et Chir., 1888.

[2] Miraillié, Gaz. des Hôp., 1893, lxvi, 889.

scirrhous type, and is composed of firm, fibrous nodules. Occasionally it is encephaloid, soft, and cellular. Colloid carcinoma has been observed.

Roux[1] described a cystic epithelioma, and Sotti a malignant adenocystoma with metastases in the lymphatic glands and lungs.

Secondary deposits are most *common in the liver*, but may be found anywhere.

A variety of carcinoma characterized by great irregularity in the size and shape of its cells, and believed to originate in the island of Langerhans, has been described by Hiller and Goodall. Secondary interstitial pancreatitis is present in some cases, and occasionally small cysts may occur.

Carcinomatous growths of the pancreas are usually of small size (5 to 10 cm. in diameter), but occasionally they are quite large. When the tumor invades adjacent organs, such as the liver, bile-passages, stomach, and duodenum, the origin of the primary growth may be in doubt.

**Effects of Adhesions and Pressure.**—Adhesions may be formed between the tumor and adjacent organs, and various lesions may result from pressure. Thus, occlusion of the duodenum or pylorus may cause dilatation of the stomach, or the stomach may be compressed against the abdominal wall, or compression of the transverse colon may cause intestinal obstruction.

*Obstruction of the bile-duct* with *dilatation of the gall-bladder is common.* Pressure on the left ureter may cause hydronephrosis, or on the portal vein may produce ascites.

Edema of the lower extremities may follow pressure on the vena cava. The tumor tissue may invade the stomach or duodenum, and perforation of these viscera may occur. The pancreas may be secondarily invaded from the stomach or duodenum.

**Symptoms.**—The symptoms of carcinoma of the pancreas are modified by the position of the growth and by the degree of chronic pancreatitis which is sometimes associated with it.

In the largest percentage of cases of cancer of the pancreas the head of the gland is involved.

The earliest symptoms are rapid loss of weight and strength, with loss of appetite, discomfort after eating, and distaste for meat. Nausea and vomiting may occur; constipation is most frequently present, though occasionally there may be diarrhea. In the earlier stages the movements may be normal. Cachexia is rapid, jaundice soon appears, and is progressive. The skin may become dark, almost black in color, or in some cases like the yellow color from gall-stones; the *gall-bladder enlarges and is readily recognizable* and the liver swells. There are intense prostration and weakness. Pain, particularly in the early stages, is at times *absent.* The writer has recently seen a large carcinoma of the outer third of the body, with no pain whatever. It may, when the tumor rapidly increases, be quite intense from pressure on the celiac ganglion or on adjacent nerve-trunks, and may radiate to the back or shoulders. Occasionally, there may be colicky pain due to occlusion of the pancreatic duct, which may resemble the colic of pancreatic lithiasis or colicky pains from pressure on the common bile-duct resembling biliary colic. The stool is pale in color after the appearance of the jaundice and bile is found in the urine. The obstruction of the common bile-duct is usually complete and the feces contain no stercobilin.

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[1] Cancer et Kystes du Pancreas, Paris, 1891.

With scirrhous cancer of the head of the pancreas often the tumor cannot be felt; in about one-fourth or one-fifth of the cases the cancerous mass is palpable, usually in the epigastric region, more rarely in the right or left hypochondrium. Usually the mass is immovable, unless lying in the tail of the pancreas. Occasionally it moves with respiration or transmits the pulsation of the aorta. There is frequently absence of free HCl in the stomach-contents, as may occur with cancer situated in other organs. This finding does not, therefore, always determine involvement of the stomach. Excessive vomiting with dilatation of the stomach may result from carcinomatous involvement of the pyloric end of that organ or of the duodenum.

Adhesions may cause similar symptoms. Occasionally, there may be no enlargement of the gall-bladder if the cystic or hepatic duct alone are involved by the growth, or if the gall-bladder be obliterated by previous inflammation. In some instances steatorrhea or *disturbances in the ratio of neutral fats and combined fatty acids* are present, and rarely undigested muscle-fibers are found in the stool. Bulky stools may occasionally be present. Alimentary glycosuria has been observed, and diabetes mellitus when there is great involvement of the pancreas, and particularly with a complicating interacinar pancreatitis. If the *tumor lie* near *the tail of the pancreas*, there is *no jaundice*.

Intestinal obstruction (chronic), even merging into the acute, may result from pressure on the transverse colon or ileum. The writer recently has seen a case of chronic intestinal obstruction caused by pressure from carcinoma of the outer third of the body of the pancreas. There was *no jaundice*. H. Haubold found this condition on operation. Ascites, swelling of the spleen, hemorrhoids, dropsy of the lower limbs, and hydronephrosis may result from pressure.

Perforation of adjacent viscera may occur. Chylous ascites due to perforation or rupture of the thoracic duct has been reported.

There is frequently a *marked hemorrhagic tendency*, particularly in the cases of *cancer of the head of the pancreas with jaundice*.

Hemorrhage may occur from the nose, mouth, stomach, intestines, and subcutaneously. Persistent bleeding or oozing is liable to occur, both at and subsequent to operation. Cammidge states the C-reaction (Cammidge) is present in about 25 per cent. of all cases and the

A-reaction in most cases. The writer is skeptical as to the value of these reactions, though still experimenting with them. Examination of the stool, the urine, and *testing the pancreatic functions* may be an aid to diagnosis.

**Diagnosis.**—Rapid emaciation, progressive weakness, increasing anemia, jaundice becoming very intense, dilatation of the gall-bladder, and epigastric tumor (not always present) are significant of carcinoma of the head of the pancreas; glycosuria and, occasionally, steatorrhea are confirmatory when present. The patient is usually over forty. The course is very rapid, not over a year, and after the onset of jaundice, only six to eight months; on distending the stomach with air or carbonic acid gas, the tumor, if present, disappears. The temperature is subnormal. Symptoms from pressure, such as anasarca, etc., occur in some cases. With cancer of the outer part of the body of the pancreas there is no jaundice and no dilatation of gall-bladder.

*Cholelithiasis.*—There is a preliminary history of gall-stone attacks; the jaundice comes on after an attack of pain and is sudden. Remissions of jaundice sometimes occur; the impacted calculi may produce intense jaundice.

Some bile usually escapes past the gall-stones into the intestines, and stercobilin is present in the stools. The bile is likely to become infected, and ague-like paroxysms occur with an irregular temperature. There is *no distended gall-bladder*, but there is a rigid right rectus muscle. There is frequently a tender point above and to the right of the umbilicus, and there may be pain which passes to the mid-scapular region or beneath the right shoulder-blade.

As a rule, the liver is not enlarged with cholelithiasis, though it may be enlarged and tender in an acute attack. The history is long.

*Chronic Pancreatitis.*—With chronic pancreatitis the *history is long*, the jaundice is, as a rule, not complete, and may be *slight* or *absent;* infection of the pancreatic duct and infective cholangitis are often present, as shown by occasional chills and temperature. The loss of flesh is less marked. Only after a long period (much *longer than one can live with cancer*) may there be anasarca or enlargement of the abdominal veins. There is a history of attacks of pain and local tenderness may be present. At operation for gall-stones, when enlargement of the head of the pancreas can be felt in a patient before middle life, or adhesions of long standing, and the history is long, the disease is apt to be chronic pancreatitis. Sometimes enlarged glands are found with this condition, but they are apt to be *discrete*, while with cancer they are usually *confluent*. The loss of flesh and strength is less marked, and the gall-bladder is *seldom distended*, though rarely it is enlarged. Anemia is less marked.

Cancer of the common bile-duct is rare and is usually associated with gall-stones. If the papilla is involved, the symptoms cannot be distinguished from cancer of the head of the pancreas. If the cancer lie above the opening of the pancreatic duct into the ampulla, the pancreatic functions will not be so markedly disturbed and the loss of flesh will not be so rapid.

*Tumor.*—Tumor of the pancreas is usually less movable than tumors of the *pylorus* or *colon*, and its position does not change, as do tumors of these organs when distended with carbonic acid gas or air. It is found in about one-fifth of the cases, and lies in the epigastrium usually, though occasionally in the right or left hypochondrium. Carcinoma of the pancreas can be at times determined by inflation of the stomach and colon, since the pancreatic tumor becomes covered by the distended organs.

*Cancer of the Liver.*—With cancer of the liver the jaundice is absent or less intense and the liver soon enlarges. Irregular nodules can be felt on the surface and edges. Cachexia is marked. In the early stages syphilis can be excluded by the Wassermann or Noguchi's tests.

*Cancer of the Pylorus.*—Cancer of the pylorus can be diagnosed by the usual methods, but sometimes both pancreas and stomach are involved. Cammidge sets great store on his urinary test as an aid to diagnosis, but the writer has not been impressed as to its value.

**Treatment.**—In the earlier stages, when doubt arises as to whether the condition be one of chronic pancreatitis or carcinoma, *exploration is indicated*, with drainage of the gall-bladder, if such be required.

Obstruction of the bowel or pyloric orifice of the stomach, when occurring, must necessarily be relieved to prolong life. When the carcinoma involves the head of the pancreas, life is not prolonged by operation to any extent, and the writer believes operative procedure to be useless. If the growth involve the tail of the gland and there are no metastases and only a few enlarged glands, a partial pancreatectomy may be indicated. So far the results of operation for carcinoma of the pancreas have been exceedingly bad, and life does not seem to be prolonged to any extent.

Medical treatment must be symptomatic, morphin for pain, if such occur. Chlorid or lactate of calcium, 60 to 90 grains (4.0–6.0) per day, for hemorrhage, pancreon tablets, liquor pancreaticus, or one of the preparations of the pancreatic ferments to aid digestion, and the usual treatment of symptoms. The bowels should be properly regulated.

### SARCOMA OF THE PANCREAS

Primary sarcoma of the pancreas is a very rare disease. Out of 11,492 autopsies Segré found this condition only twice, and Hale White only one case in 6708 autopsies. Kakels[1] collected 21 cases, among which 10 only were believed to be primary.

Fibrosarcoma, medullary sarcoma, mixed-cell sarcoma, lymphosarcoma, spindle-celled sarcoma, and angiosarcoma have been described. Sarcomatous degeneration of an ecchinococcus cyst of the tail of the pancreas has also been reported successfully removed by operation.

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[1] Amer. Jour. Med. Sci., 1902, cxxiii, 471.

**Age of the Patient.**—The age of the patients vary from four to seventy years. The period of life may, therefore, not be significant for diagnosis.

**Symptoms.**—Rapid loss of flesh and strength, and the symptoms described under Carcinoma may occur.

Secondary sarcoma of the pancreas is not uncommon. It occurs most frequently as a lymphosarcoma arising from a primary sarcoma of the duodenum, mediastinum, or abdominal lymph-glands. Secondary melanotic sarcoma of the pancreas has also been described, the primary growth being situated in the eye.

**Diagnosis.**—The general course of the disease resembles carcinoma of the pancreas, though the tumor may grow more rapidly.

**Treatment.**—The medical treatment should be symptomatic. Surgical procedure results unfavorably, though exploration for diagnosis may be necessary.

### Adenoma of the Pancreas

Cystic adenomata have been described. Simple adenoma may originate from the epithelium of the duct, the gland acini, or an island of Langerhans. Fibro-adenoma has been reported. Nicholls describes a tumor consisting of a stroma of connective tissue arranged in irregular alveoli which contained cells of a glandular type, probably arising from an island of Langerhans. One case of fibro-adenoma of the head of the pancreas has been reported by Biondi,[1] which was palpable and caused jaundice. Recovery followed removal. Small tawny, yellow tumors have been described which histologically resembled an island of Langerhans.

**Symptoms.**—No characteristic symptoms are produced except from pressure on the ducts or adjacent organs, and the presence of a palpable tumor.

**Treatment.**—Attempt should be made to extirpate the tumor by operation.

**Lymphadenoma.**—Lymphadenoma of the pancreas is extremely rare. Only 2 cases are referred to, which were met with in patients who died from Hodgkin's disease.

[1] Riforma Med., 1896, ii, 97.

## CHAPTER XLIII

# DEGENERATIVE CHANGES OF THE PANCREAS—THE PANCREAS AND DIABETES—HEMOCHROMATOSIS

### DEGENERATIVE CHANGES OF THE PANCREAS

Degenerative changes occurring in the liver, kidneys, and other parenchymatous organs may also affect the pancreas. Like other organs, the pancreas may diminish in size and weight as the years advance, or the same condition may result from chronic diseases or from marasmus; senile atrophy is accompanied in many instances by sclerotic changes in the blood-vessels, or it may be the result of malnutrition, such as occurs with chronic wasting diseases.

Atrophy of the pancreas is met with in a considerable number of diabetics, and the cause of both conditions is usually a chronic interstitial pancreatitis.

Opie shows there are cases of diabetes in which the pancreas were markedly diminished in size, though no changes could be observed in the structure of the glands. Possibly this condition is *congenital*, and at some period of life the pancreas is so small that it cannot fulfil the demands made upon it, so diabetes results. Another condition believed to be due to congenital pancreatic deficiency was first described by Byron Bramwell as "pancreatic infantilism." Secondary atrophic changes may occur in the pancreas as the result of pressure from aneurysm, and new growths or chronic interstitial inflammation which accompanies pancreatic calculi, pancreatic cysts, hemorrhage, or abscess.

The pancreas may undergo fatty, hyaline, or amyloid degeneration, and focal necrosis.

**Fatty Degeneration.**—*Etiology.*—The prolonged use of alcohol usually produces an excess of fat or fatty degeneration in the islands of Langerhans. Fatty degeneration is caused most *frequently* by inflammation of the pancreas, but it may occur with infectious diseases, toxemias, or from poisoning by phosphorus or the mineral acids, or be associated with pancreatic lithiasis.

In extreme cases the entire organ may be transformed into a mass of fatty tissue, and the pancreas is yellow or yellowish white, soft, and larger than normal. On section the gland is lobulated and consists of masses of fat, separated by strands of fibrous tissue in which the remains of the larger ducts and remnants of the gland structure are embedded. The epithelium contains numerous fat globules and the interstitial tissue is edematous.

**Amyloid Degeneration.**—Amyloid degeneration occurs with a similar condition in other organs, and is associated with the same causes as the cachectic state, particularly when due to prolonged suppuration; tuberculous bone diseases; less frequently pulmonary tuberculosis; tertiary syphilis; the cancerous cachexia; rickets; protracted convalescence from acute infectious diseases; and chronic enterocolitis. It does not attain the severity such as is seen in the liver, spleen, and kidneys.

The application of iodin to the pancreas turns the area of amyloid degeneration a dark brown.

Metachromatic stains, such as gentian violet or methyl green, may be used to demonstrate the amyloid condition.

**Hyaline Degeneration.**—This form of degeneration exhibits a *special tendency to attack the interacinar islands of Langerhans*, and generally leaves the secreting parenchyma unaffected. *Diabetes is particularly associated with it.*

*Pathology.*—There is first an increase in size of the cells of the islands of Langerhans and an alteration of their protoplasm. The nuclei disappear with the death of the cells and the cell protoplasm finally becomes homogeneous. Small masses of hyaline matter fuse together and form larger collections.

The hyaline material may occupy nearly the entire area of the island and only a few epithelial cells be present. The island appears as a circumscribed hyaline structure of homogeneous material in a series of broken, twisted columns, between which lie capillary walls. The lumen of the blood-vessels is patent and red blood-corpuscles are visible between the hyaline masses.

It is *usually* accompanied by chronic interacinar pancreatitis, though the increase in interstitial tissue is sometimes slight. Arteriosclerosis has been present in a number of cases. The hyaline material stains deeply with eosin, picric acid, and other acid dyes, and shows little affinity to nuclear stains. It does not give the amyloid reaction. Similar hyaline degeneration is not present in the other organs.

**Focal Necrosis.**—In a case of diabetes reported by Opie there was present a lesion of the pancreas, resembling the focal coagulation necrosis observed in the liver in typhoid fever and like infections. It affected both the islands of Langerhans and also the parenchyma. The process is acute, but there is a commencing chronic proliferation of interstitial tissue replacing the defects caused by destruction of the cells. Focal necrosis of the pancreas has been found in a number of cases associated with lobar pneumonia and other infections.

## THE PANCREAS AND DIABETES

The writer has already referred to the fact that the islands of Langerhans produce an internal secretion which influences carbohydrate metabolism, and disease of the islands will produce diabetes mellitus. Opie[1] reports findings in 288 cases of diabetes mellitus: interacinar pancreatitis in 43.4 per cent. (125 cases); interlobular pancreatitis (13 cases); atrophy (65 cases); lipomatosis (18 cases); while calculi, cyst, carcinoma, and focal necrosis were found in a few others. There were a few cases with lesions of the islands of Langerhans, such as hyaline degeneration, sclerosis, adenoma, and hypertrophy—with normal parenchyma. There were 34 cases of diabetes with a normal pancreas and 5 cases with normal parenchyma, but the islands reduced in number.

Cecil[2] found lesions of the islands of Langerhans in 79 (88 per cent.) of 90 cases of diabetes.

The changes which destroy the islands of Langerhans, especially interacinar pancreatitis and hyaline degeneration, are almost constantly accompanied by diabetes, while the lesions which destroy the parenchyma and invade the islands of Langerhans only when the lesion is far advanced, such as in the case of interlobular pancreatitis, pancreatic calculi, and carcinoma, are usually not accompanied by diabetes. Acute lesions of the pancreas, such as acute pancreatitis (hemorrhagic necrosis) or suppuration, rarely cause glycosuria, since usually sufficiently healthy parenchyma remains to prevent its onset.

In a small percentage of cases of diabetes mellitus (6.6 per cent.) of those reported by Cecil no abnormality of the pancreas was found. Changes in the central nervous system, the liver, and the kidneys have been followed by glycosuria, and even by diabetes, and diabetes has also accompanied exophthalmic goiter and acromegaly.

Arteriosclerosis is present in a large number of diabetics due to its production of interstitial pancreatitis. The close association between cirrhosis of the liver and diabetes has often been observed, the latter in the majority of cases the result of chronic interstitial pancreatitis, both the cirrhosis and pancreatitis usually being the result of the same factor, alcohol, for example.

Higgins[3] and Ogden found glycosuria in 9.3 per cent. of 212 cases of traumatism affecting the head, and in 21.9 per cent. out of 45 cases of fractured skull. Diabetes is occasionally associated with tabes or with multiple sclerosis or tumors of the medulla.

A renal diabetes has been suspected, and Klemperer[4] has described a case of diabetes associated with nephritis.

Among other conditions with which diabetes has been associated are myxedema, disease of the adrenals, and acromegaly. Glycosuria has been observed to follow the use of thyroid extract, and it has been suggested that glycosuria occurring with exophthalmic goiter is referable to abnormal activity of the thyroid.

In many cases diabetes, which is associated with arteriosclerosis, cirrhosis of the liver, hemochromatosis, Graves' disease, and acromegaly, is secondary to a pancreatitis which accompanies these diseases.

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[1] Disease of the Pancreas.

[2] Ibid.

[3] Boston Med. and Surg. Jour., 1895, cxxxii, 197.

[4] Berliner Klin. Woch., 1896, xxxiii, 571.

In about 12 per cent. of all cases diabetes is *unaccompanied* by a lesion of the islands of Langerhans, and diabetes *with a normal pancreas* usually occurs during the early *period of life*, most frequently before the age of thirty.

Diabetes which occurs during middle life and later can generally be referred to a lesion of the pancreas. Interacinar pancreatitis is responsible for about two-thirds of the cases of diabetes after forty years of age. Though alimentary glycosuria may occur without pancreatic disease, with hysteria and other necroses, from the administration of excessive sugar or starchy products, etc., its *persistence* suggests the existence of a pancreatic lesion.

## HEMOCHROMATOSIS

Von Recklinghausen,[1] under the term "hemochromatosis," describes a condition of pigmentation affecting various organs. He believes the brown pigment is derived from the hemoglobin of the blood. There is an iron-containing pigment, "hemosiderin," in the epithelial cells of the various glands, chiefly the liver and pancreas. There is also an iron-free pigment, "hemofusan," in the muscle-cells of the gastro-intestinal tract, also in the muscle-cells of the blood and lymph-vessels, and connective-tissue cells. Cirrhosis of the liver is associated with the pigmentation.

**Bronzed Diabetes.**—Hanot[2] and Chauffard describe a closely related condition of diabetes mellitus associated with hypertrophic cirrhosis of the liver and bronze-like pigmentation of the skin, "diabète bronzé."

The diabetes is rapidly fatal. Associated with it is an hypertrophic cirrhosis of the liver, and bronzing of the skin is present in the majority of cases.

*Etiology.*—The pigment deposited in the liver and other organs is undoubtedly derived from the hemoglobin in the blood. In many cases it has been accompanied by conditions causing an active destruction of the red blood-cells, such as morbus maculosus, the hemorrhagic diathesis, etc.

It is believed that phagocytosis of the red blood-corpuscles by the parenchymatous cells of various organs takes part in the process. Chronic interstitial pancreatitis of the interacinar variety has been reported in these cases, as well as hypertrophic cirrhosis of the liver.

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[1] Tagebl. d. 62 Versamml. deutsch. Naturforsher v. Aertze in Heidelberg, 1889, 324.

[2] Rev. de Méd., 1882, ii, 385.

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